A DISSERTATION ON

"OCULAR MANIFESTATIONS IN CHILDREN WITH DELAYED MILESTONES-A CLINICAL STUDY"

Submitted to

THE TAMIL NADU DR. M. G. R. MEDICAL UNIVERSITY

In partial fulfilment of the requirements

For the award of degree of

M.S. (Branch III) --- OPHTHALMOLOGY

GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMIL NADU

MAY 2018

CERTIFICATE

This is to certify that the study entitled **"OCULAR MANIFESTATIONS IN CHILDREN WITH DELAYED MILESTONES- A CLINICAL STUDY"** is the result of original work carried out by **DR.SOUNDARYA.B**, under my supervision and guidance at **GOVT. STANLEY MEDICAL COLLEGE, CHENNAI.** The thesis is submitted by the candidate in partial fulfilment of the requirements for the award of M.S Degree in Ophthalmology, course from 2015 to 2018 at Govt.Stanley Medical College, Chennai.

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DECLARATION

I hereby declare that this dissertation entitled "**OCULAR MANIFESTATIONS IN CHILDREN WITH DELAYED MILESTONES- A CLINICAL STUDY**" is a bonafide and genuine research work carried out by me under the guidance of **PROF. DR.B.RADHAKRISHNAN M.S. D.O.,** Unit chief and Head of the Department, Department of Ophthalmology, Government Stanley Medical college and Hospital, Chennai – 600001.

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Place:Chennai Dr. Soundarya.B

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INSTITUTIONAL ETHICAL COMMITTEE,
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Title of the Work : Ocular manifestation in Children with delayed milestones A Clinical study.

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The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 02.12.2016 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

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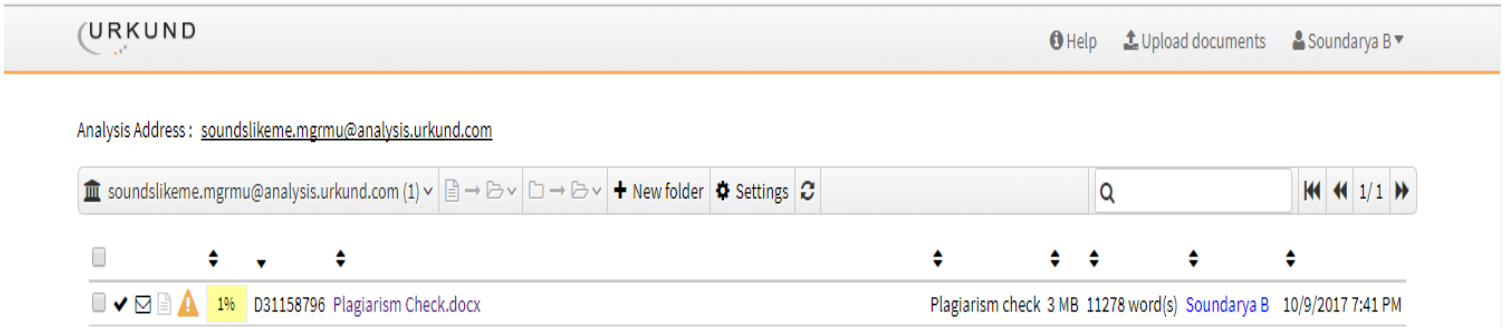
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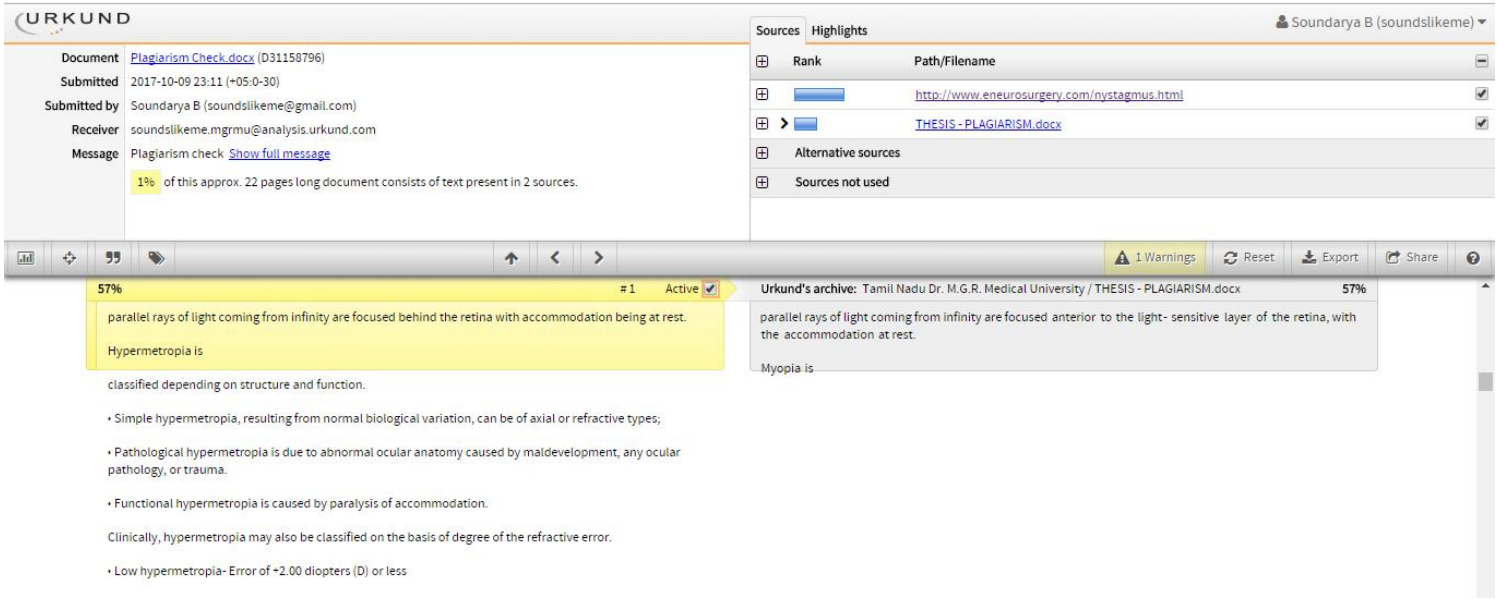
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parallel rays of light coming from infinity are focused behind the retina with accommodation being at rest.
Hypermetropia is
classified depending on structure and function.
• Simple hypermetropia, resulting from normal biological variation, can be of axial or refractive types;
• Pathological hypermetropia is due to abnormal ocular anatomy caused by maldevelopment, any ocular pathology, or trauma.
• Functional hypermetropia is caused by paralysis of accommodation.
Clinically, hypermetropia may also be classified on the basis of degree of the refractive error.
• Low hypermetropia- Error of +2.00 diopters (D) or less
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parallel rays of light coming from infinity are focused anterior to the light- sensitive layer of the retina, with the accommodation at rest.
Myopia is

CERTIFICATE - II

This is to certify that this dissertation work titled "OCULAR MANIFESTATIONS IN CHILDREN WITH DELAYED MILESTONES"

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CONTENTS

PART-I

INTRODUCTION

"A significant delay in two or more developmental domains" is termed as developmental delay[1]. The developmental domains included in the definition are:

- Gross motor
- Fine motor
- Speech/ language/hearing
- Personal/social

Significant delay is said to be present when development or ability is two standard deviations or more below the mean on accepted developmental testing like Denver Developmental Screening Test.

The prevalence of development delay in India is found to be about 2.5% of paediatric population under 5 years.

There are a number of studies that have analyzed the risk factors for developmental delays and emphasised early detection and screening for these delays.

Various factors like maternal, genetic, perinatal, post-natal and social factors play a role in impairment of development.

There is notable evidence reporting higher incidence of visual abnormalities among children with mental and developmental delays. The ocular signs are most often missed, as handicaps in other functional domains are coexistent.

Visual impairment retards or changes ocular as well as the general development of the child. A number of research studies have put forward the fact that there is pronounced influence of

numerous forms of sensory deprivations on the ocular and visual development. The effect of visual deprivation also plays a major role in adequate development of other domains, especially from the social aspect.

The importance of visual function in toddlers is emphasized, because their visual system is yet to develop completely, thus putting them at risk of evolving amblyopia from either uncorrected high ametropia or anisometropia. Visual distress may progress to cause lifelong visual handicap.

NEED FOR THE STUDY

The load of visual handicap in children, particularly in a child with delayed milestones, is of towering importance, owing to the lifelong impact of the visual impediment on the development of other functions.

Prompt diagnosis of the issue may accelerate treatment or any other modality of management when the disability or problem is not treatable.

The attention that ocular deficits are given is usually lower than necessary, and it has been noted that certain abnormalities frequently go untreated due to missed diagnosis or lack of awareness of their value in children with developmental delay.

The parent or guardian also does not note a visual disability readily in such children, as inattention of the child to his/her surroundings is attributed to delays in other domains.

Thus, this study was done to assess the prevalence of ocular manifestations, as a component of ophthalmological evaluation for children referred from paediatric neurology department of our hospital, for the betterment of academic achievement and social skills in the susceptible early years of life.

REVIEW OF LITERATURE

- **The Denver development scale** for evaluating developmental delay was developed by William K. Frankenburg and first introduced by him and Josiah.B. Dobbs in 1967.

- **Kauai Longitudinal Study**, Hawaii, 1955, showed that factors like low birth weight, prematurity, birth injury and other social factors like poverty played an important part in development of serious learning or behaviour problems.

- **Morgan** (1977), **Ackroyd et al**. (1974), **Fine et al**. (2002), **Valvo** (1968), and **Gregory and Wallace** (1963) did various studies on effects of early bilateral visual deprivation.

- **Mitchell et al.,** 1984 **and Murphy and Mitchell,** 1987 studied the effects of mono ocular visual deprivation and effect of patching therapy in amblyopia.

- **Accardo et al**. 2008 and **Lipkin & Schertz**, 2008 stated that "throughout the development, fine motor skills merge with visual skills, allowing the assessment of problem solving based on visual-motor skills, an essential function of intellectual development".

- Regarding visual acuity, **Lewis and Maurer** suggested that grating acuity becomes adult-like by 4-6 years and letter acuity by 6 years.

- **Daw** stated that adult-like acuity of 30 cycles per degree (~6/6) is reached by 3 years, and clinically, it is assumed that VA is same as adult values of 6/6 by 5 years.

- **The Forced Preferential Looking technique** of visual acuity assessment in children was conceived by Davida Teller.

- **Visually Evoked Potentials** initiated by a flash were noticed in the initial years of clinical encephalography (EEG) in the 1930s. Dawson was the first one to demonstrate a signal-averaging device in 1951 and signal-averaging computers have then been available since 1960s.

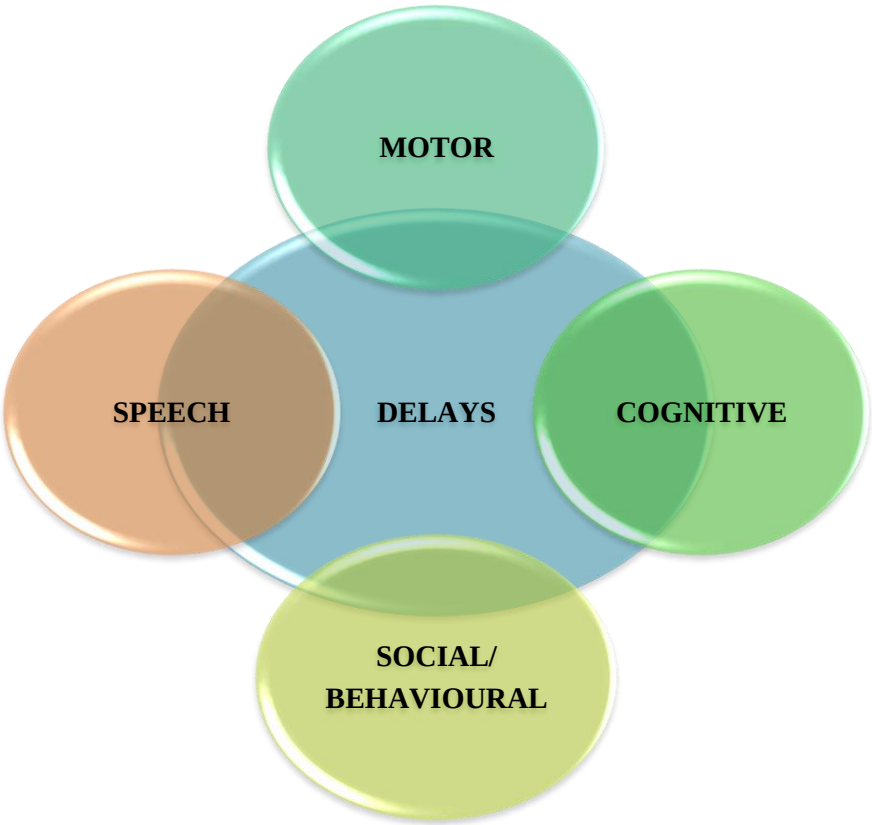
- **Akinci A et al** studied the ocular manifestations in children with intellectual disabilities and reported that 77% of the children had ocular manifestations.

- **Mets M B et al** studied "causes of childhood blindness and visual loss in an institution for severely mentally retarded children" and concluded that bilateral optic atrophy was the most common cause of visual loss.

DEVELOPMENTAL DELAY

Delay in development can have a drastic effect on various skills of a child. It can involve physical, intellectual, communicative, emotional or social domains. The delays frequently alter more than one developmental sector of the child. When a child manifests lags in most or all of these domains, it is known as **global developmental delay.**

A detectable cause can be identified in some of these cases. However, major proportions of the children do not have an evident causative factor for the delay, or multiple delays.



MOTOR DELAYS

Delays in this domain impede the child's ability to coordinate large muscle groups-gross motor functions, and smaller muscle groups-fine motor functions. Infants with gross motor delays usually present with difficulty rolling over or crawling. Toddlers with gross motor delay may seem clumsy or have trouble walking up and down stairs, whereas children with fine motor delays may show difficulty in holding small objects, like toys, or doing tasks like tying shoes or brushing teeth.

Motor delays can also be due to genetic conditions, like achondroplasia, that causes shortening of the limbs, or due to conditions like cerebral palsy or muscular dystrophy which affect the muscles. These delays may also be due to structural problems, like limd length discrepancy.

COGNITIVE DELAYS

Cognitive delays may alter the intellectual behaviour of a child, thus hampering with awareness and leading to difficulty in learning and comprehension that frequently becomes obvious after a child begins schooling. These children with cognitive delays may also have difficulty interacting and playing with other children of their age group.

Cognitive delays may occur in children who have had cerebral injury due to infections-meningitis or encephalitis. Shaken baby syndrome (Abusive brain trauma), chromosomal disorders like Down's syndrome and epilepsy, may also raise the risk of a cognitive delay. A clear cause, however, is not established in maximum cases.

BEHAVIOURAL, SOCIAL AND EMOTIONAL DELAYS

Behavioural, Social and Emotional Delays often accompany Developmental delays that some children have (developmental delays can include related neurobehavioral disorders like attention deficit hyperactivity disorder and autism spectrum disorder). These children may react to their surrounding in a different way or process data in a different way as compared to others of same age. This is due to brain development differences. This affects a child's capability to communicate, learn and interact with others.

Developmental delays in some children, often causes the children to struggle with emotional and social skills. Some examples include:

- Difficulty comprehending social prompts
- Starting a communication with other people
- Continuing a conversation
- Trouble dealing with the feeling of being upset or annoyed
- Handling variation or change

When the situation is very emotionally or socially difficult, these children could exhibit lengthy irritabilities and take longer than others to relax. This conduct can be a sign that a child needs more care by altering the environment or learning skills to handle these emotional and social difficulties.

DELAYED SPEECH

Commonly, a child with Speech delays has a combination of both the below types:

- Receptive Language Disorder - Child has trouble comprehending concepts or words. This type of delay causes difficulty identifying body parts, shapes or colours
- Expressive Language Disorders - vocabulary of complex sentences and words is reduced compared to other children of the same age. This type of delay causes the child to be slow to start babbling, talking or creating sentences

Speech Production disorder is when a child has an oral motor issue (example – weak mouth muscles or trouble to move the jaw or tongue—that affects speech production)

Causes:

- Physiological causes –genetic conditions, brain damage or hearing loss
- Environmental factors - lack of stimulation

However, in many cases, the cause seems to be unknown.

EVALUATION OF DEVELOPMENTAL DELAY

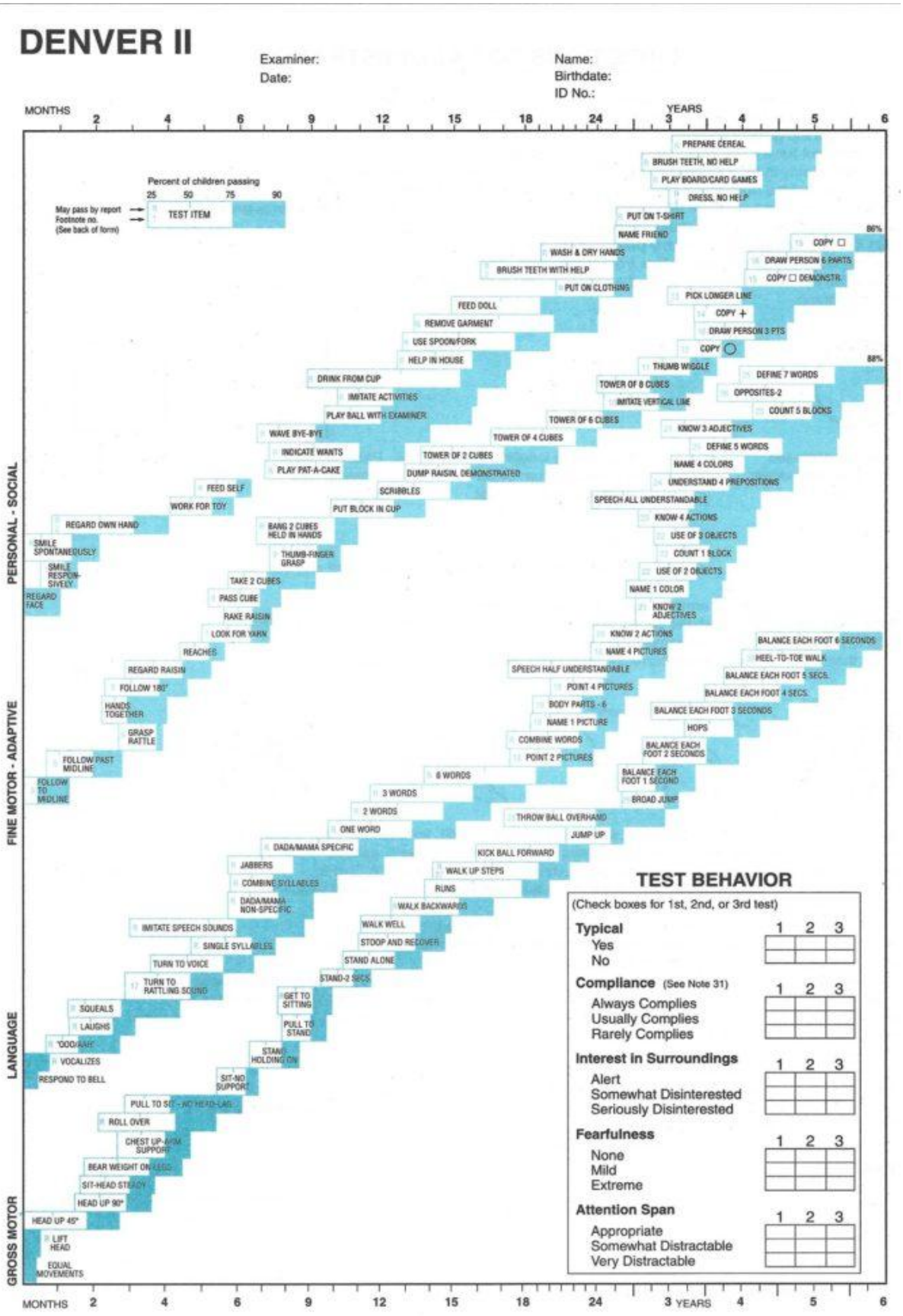
THE DENVER DEVELOPMENTAL SCREENING TEST:

It is made up of 125 components , that are divided into four parts:

1. Gross motor
2. Fine motor
3. Language
4. Social/Personal

Every individual component is selected suitably in accordance to the age of the child and scoring is done objectively as- "pass", "fail", "refusal" (uncooperative), or "no opportunity" (if test was not performed).

- **“Pass”** indicates that the child is able to perform the skill or task that is usually performed by 90% of the group of children younger than this child.
- **“Fail”** is divided into-
    - “Delayed"- if the child fails a test that has been acceptably done by 90% of younger children
    - "Caution"- if the child fails or refuses a test that 75 to 90% of younger children have adequately passed.


DENVER II
Examiner:
Date:
Name:
Birthdate:
ID No.:
MONTHS
YEARS
Percent of children passing
25 50 75 90
May pass by report
Footnote no.
(See back of form)
TEST ITEM
PERSONAL - SOCIAL
FINE MOTOR - ADAPTIVE
LANGUAGE
GROSS MOTOR
PREPARE CEREAL
BRUSH TEETH, NO HELP
PLAY BOARD/CARD GAMES
DRESS, NO HELP
PUT ON T-SHIRT
NAME FRIEND
WASH & DRY HANDS
BRUSH TEETH WITH HELP
PUT ON CLOTHING
FEED DOLL
REMOVE GARMENT
USE SPOON/FORK
HELP IN HOUSE
DRINK FROM CUP
IMITATE ACTIVITIES
PLAY BALL WITH EXAMINER
WAVE BYE-BYE
INDICATE WANTS
PLAY PAT-A-CAKE
FEED SELF
WORK FOR TOY
REGARD OWN HAND
SMILE SPONTANEOUSLY
SMILE RESPONSIVELY
REGARD FACE
BANG 2 CUBES HELD IN HANDS
THUMB-FINGER GRASP
TAKE 2 CUBES
PASS CUBE
RAKE RAISIN
LOOK FOR YARN
REACHES
REGARD RAISIN
FOLLOW 180°
HANDS TOGETHER
GRASP RATTLE
FOLLOW PAST MIDLINE
FOLLOW TO MIDLINE
TOWER OF 2 CUBES
DUMP RAISIN, DEMONSTRATED
SCRIBBLES
PUT BLOCK IN CUP
TOWER OF 4 CUBES
TOWER OF 6 CUBES
TOWER OF 8 CUBES
IMITATE VERTICAL LINE
THUMB WIGGLE
COPY ○
DRAW PERSON 3 PTS
COPY +
PICK LONGER LINE
COPY □ DEMONSTR.
DRAW PERSON 6 PARTS
COPY □
80%
DEFINE 7 WORDS
OPPOSITES-2
COUNT 5 BLOCKS
KNOW 3 ADJECTIVES
DEFINE 5 WORDS
NAME 4 COLORS
UNDERSTAND 4 PREPOSITIONS
SPEECH ALL UNDERSTANDABLE
KNOW 4 ACTIONS
USE OF 3 OBJECTS
COUNT 1 BLOCK
USE OF 2 OBJECTS
NAME 1 COLOR
KNOW 2 ADJECTIVES
KNOW 2 ACTIONS
NAME 4 PICTURES
SPEECH HALF UNDERSTANDABLE
POINT 4 PICTURES
BODY PARTS - 6
NAME 1 PICTURE
COMBINE WORDS
POINT 2 PICTURES
6 WORDS
3 WORDS
2 WORDS
ONE WORD
DADA/MAMA SPECIFIC
JABBERS
COMBINE SYLLABLES
DADA/MAMA NON-SPECIFIC
IMITATE SPEECH SOUNDS
SINGLE SYLLABLES
TURN TO VOICE
TURN TO RATTLING SOUND
SQUEALS
LAUGHS
"OOO/AAH"
VOCALIZES
RESPOND TO BELL
BALANCE EACH FOOT 6 SECONDS
HEEL-TO-TOE WALK
BALANCE EACH FOOT 5 SECS.
BALANCE EACH FOOT 4 SECS.
BALANCE EACH FOOT 3 SECONDS
HOPS
BALANCE EACH FOOT 2 SECONDS
BALANCE EACH FOOT 1 SECOND
BROAD JUMP
THROW BALL OVERHAND
JUMP UP
KICK BALL FORWARD
WALK UP STEPS
RUNS
WALK BACKWARDS
WALK WELL
STOOP AND RECOVER
STAND ALONE
STAND-2 SECS.
GET TO SITTING
PULL TO STAND
STAND HOLDING ON
SIT-NO SUPPORT
PULL TO SIT - NO HEAD-LAG
ROLL OVER
CHEST UP-AVM SUPPORT
BEAR WEIGHT ON LEGS
SIT-HEAD STEADY
HEAD UP 90°
HEAD UP 45°
LIFT HEAD
EQUAL MOVEMENTS
TEST BEHAVIOR
(Check boxes for 1st, 2nd, or 3rd test)
Typical
Yes
No
Compliance (See Note 31)
Always Complies
Usually Complies
Rarely Complies
Interest in Surroundings
Alert
Somewhat Disinterested
Seriously Disinterested
Fearfulness
None
Mild
Extreme
Attention Span
Appropriate
Somewhat Distractable
Very Distractable
1 2 3

DEVELOPMENT OF VISION

Considering all cognitive functions, vision acts as a major factor in development of intercommunication and bonding, “spatial awareness”, oculomotor and other motor functions.

- Development is completed to a large extent structurally by the age of 2-3 years, but functionally the changes continue to occur throughout life.
- Vision increases rapidly up to one year of age, following which it matures in a more gradual pace to attain adult levels at around 5-6 years of life.
- The central cones begin to function by term. However visual acuity does not reach 6/6 until 6 months to 30 months.
- Causes for this lag include the insufficient development and specialization of photoreceptors, synaptic maturation of the inner layers of retina, and upper visual pathway myelination.
- The adult appearance of the foveal cones is not attained till 4months after term, and myelination of visual pathway continues till 2 years of life.

VISUAL DEVELOPMENT INVENTORY

VISUAL DEVELOPMENT INVENTORY	Newborn	1 Month	2 Months	4 Months	6 Months	9 Months	12 Months	18 Months	24 Months	3 Years
OCULOMOTOR										
Fixation	To face →									
Saccades										
H	⊕ →								One shift	
V	⊖	⊖	⊕ →						to target	
Pursuits	⊖	⊖	Emerging	⊕ →						
Visually directed reaching	⊖	⊖	⊖	?	⊕ →					
Face regard	⊕ →									
OKN: T-N and N-T response	Asymmetric ⊕ →	Asymmetric	Asymmetric	Asymmetric	Symmetric →					
ACUITY	20/400	20/300	20/150		20/50			20/40	20/30	20/20
Preferential	to	to	to →		to →		→	to	to	
Looking OU	20/1200	20/1200	20/600		20/200			20/100	20/80	
Visual Evoked Response VEP OU	20/100	20/200	20/80		20/40					
BINOCULARITY								Adultlike levels		
Alignment		⊕ →					→	of Angle Lambda →		
Near point of convergence	Up to 10 in →			→	To nose →					
Fixation of moving target	⊖	⊖		Response ⊕ →						
10 Δ response	⊖	⊖			70% of time →					
Stereopsis	None			Emerging	Well developed					
ACCOMMODATION				Well developed						
Accuracy	Accurate for 30 cm (12 in)				Accurate for 75 to 150 cm →					
Lag				+0.75 →						
PUPIL RESPONSES	⊕Sluggish	Well developed →								
Color Vision	Notices color Can't distinguish R, G, or Y		Distinguishes R-G not Y-G	Recognizes similar hues within color group						Normal
Blink response to visual threat			⊕ →							
Contrast sensitivity function			Adult-like low frequency attenuation							Adult-like btwn 3-5 yr

Key: ⊕ Emerging; ⊖ Not existing; → Continues; R = Red; G = Green; Y = Yellow

(Courtesy of Dr. Janice Scharre, Illinois College of Optometry, Chicago.)

VISUAL MILESTONES:

- **Following birth-** Fixes and follows light source/face/ large sized toy that is colourful.
- **At 1 month**-

    1. Central fixation which is steady and maintained
    2. Ability to follow a slow moving target
    3. Convergence
    4. Face preference

- **At 3 months**- Binocular vision with eye coordination, follows moving light/follows face, responsive to smile.
- **At 6 months**-Reaches out for toys precisely.
- **At 9 months**- Searches for hidden toys.
- **At 2 years**- Matching of pictures
- **At 3 years**- Matching of individual letters (For eg., Sheridan Gardiner)
- **At 5 years**- Snellen chart-matching/naming

Development of ocular reflexes:

The visual reflexes which begin as primitive reflexes in-utero, continue to develop as the infant grows. The reflexes seen in the various months are as follows:

Age	Reflex
Birth	Blinking (to light stimulus)
1 week	Vestibulo-ocular
2 weeks	Small saccades
2 months	Large saccades Pursuit Bifoveal fixation Convergence
3 Months	Uniocular fixation
4 months	Fusional vergence Sensory fusion Stereopsis
6 months	Accommodation

DEVELOPMENTAL DELAY AND VISION

The development of vision, which is a very complicated maturation process that involves anatomical and functional ocular and CNS alterations.

Developmental delay may be associated with a delay in visual maturation in which children do not attain fixation for upto 6-12 months, following which they may exhibit normal visual functions.

They may present with completely normal ocular findings except for poor fixation because of the delay in visual system maturation. In such children, supportive therapy and reassurance is sufficient until the visual attention is achieved.

The clinical evaluation and management of vision deficit in developmentally challenged children is a complex process, posing difficulty to the physician. One such abnormality is amblyopia, which is not being adequately evaluated in these children. It can be prevented if diagnosed earlier, but this becomes difficult in these children, as precise diagnosis is not possible on a single examination at all times.

Squint and refractive errors comprise the highest percentage of the ocular manifestations. Thus it warrants the necessity for early diagnosis, developing tools for special education, and evolving techniques to prevent the progression of these defects. The incidence of squint and refractive errors has been found to be around 50% from different research works. Both these disorders are treatable to some level.

The other ocular manifestations seen in association with delayed milestones include-

- Cataract
- Optic Atrophy
- Nystagmus
- Cortical Visual Impairment (CVI)

VISION AND ITS INFLUENCE ON OTHER AREAS OF DEVELOPMENT-

The function of sight combines all other senses and thus any gross impairment leads to a constraint in most domains of development in early childhood. This restriction is directly proportional to the amount of visual loss.

Visual impairment has an influence on the development of:

- "Spatial awareness", posture and mobility
- Hand usage and fine motor coordination
- Early conceptual ability development
- Localising sound in a given space
- Perception of words and thus speech or language development
- Ability to interact and communicate in a society
- Self-care.

Development may be delayed by as late as 2 years of age. This lag can be overcome as the child reaches school age.

However, in children with profound visual impairment (perception of light only), studies have analysed that around 30% of these children are at a higher risk of stoppage or retrogression of intellectual development at two years of life.

Impaired social interaction is most evident in this category and the increased level of “autistic spectrum disorder” in blind children is well documented. The same sequel is seen even in a child who has a primary visual abnormality without noticable risk factors for cerebral damage.

PRETERM BIRTH AS A RISK FACTOR FOR VISUAL IMPAIRMENT-

Children who are born preterm have a greater risk of brain injury. The most common cerebral lesion is periventricular leukomalacia (PVL). Motor and cognitive delays are more often present in these children. Less often, there is also a risk of ocular and visual impairments, which are not as well perceived as other abnormalities. The visual impairment in these children is reported to be between 1%-3%.

Causative factors include cerebral visual impairment and retinopathy of prematurity (ROP). Refractive errors have seen to be four times higher in preterm children than those born at term. These visual abnormalities are seen as a result of ‘premature exteriorisation’ of the developing ocular system and due to the systemic sequelae of preterm birth.

As we know, hypermetropia is the most prevalent refractive error in children. In preterm children, however, myopia is seen to be more common, presenting in those children with or without history of ROP. In cases of squint, esotropia and exotropia are seen equally, as opposed to full-term children, where esotropia is three times more common than exotropia.

Children may also present with a difficulty in higher-degree processing of visual stimuli, especially in those with periventricular leukomalacia (PVL). However, it is to be noted that PVL is not a key factor, with the visual affection being a result of some other factors of prematurity, as suggested by some studies.

OCULAR MANIFESTATIONS IN DEVELOPMENTAL DELAY

REFRACTIVE ERRORS

The ocular system engages an active process of emmetropization, which involves active structural changes and leads to a coordinated growth of the refractive components, that helps in achieving the final goal of emmetropia[7]. In the initial three years of life, the cornea and lens alter structurally to counterbalance around 20 diopter increase in axial length of the growing eye. Between 3 and 13 years of age, the lens and or cornea need to alter about 3 diopters to maintain emmetropia.

MYOPIA

Here, parallel rays of light coming from infinity are focused in front of the retina with accommodation being at rest.

Myopia is seen with high heritability, thus suggesting a significant genetic component to account for the variance in the population. Multiple myopia genetic loci have been studied, placing myopia under a common complex disorder.

Myopia is classified according to amount of error as follows:

- **Low myopia**- Myopia of less than −3.00 dioptres
- **Moderate myopia**- Myopia between −3.00 and −6.00 dioptres
- **High myopia**- Myopia of more than −6.00 dioptres.

Myopia is classified in as:

i) Simple

ii) Pathological

Simple Myopia is a non progressive condition, beyond the amount that is within normal development. Visual acuity is usually good.

Whereas, **pathological myopia** is a degenerative condition associated with changes in the posterior segment of the eye, along with increase in length of antero-posterior axis of the globe. Besides axial pathological myopia, other types are due to defects in the curvature of cornea and lens or trauma or due to change in refractive index of the lens.

The pathological myopia is associated with degenerative changes in the posterior pole. It may present as an independent developmental (congenital) condition or may occur in association with other ocular disorders or systemic disease.

Congenital myopias most often remain stationary. Some may progress to produce retinal detachment. The congenital anomalies associated with myopia include microphthalmos, microcornea, microphakia, ectopia lentis. Many tapeto-retinal dystrophies are usually seen with high myopia.

HYPERMETROPIA

Here, parallel rays of light coming from infinity are focused behind the retina with accommodation being at rest.

Hypermetropia is classified depending on structure and function.

• Simple hypermetropia, resulting from normal biological variation, can be of axial or refractive types;

• Pathological hypermetropia is due to abnormal ocular anatomy caused by maldevelopment, any ocular pathology, or trauma.

• Functional hypermetropia is caused by paralysis of accommodation.

Clinically, hypermetropia may also be classified on the basis of degree of the refractive error.

• **Low hypermetropia**- Error of +2.00 diopters (D) or less

• **Moderate hypermetropia**- Error from +2.25 to +5.00 D

• **High hypermetropia**- Error over +5.00 D

Another classification is based on the results of noncycloplegic and cycloplegic (1.0% Cyclopentolate) refractions, taking the accommodative system into consideration:

•**Manifest hypermetropia**- The amount of refractive error that is brought out by noncycloplegic refraction

• **Latent hypermetropia**- The amount of refractive error that can be detected only by cycloplegia, and is overcome by accommodation in noncycloplegic conditions.

The sum of latent and manifest hypermetropia is the magnitude of hypermetropia

Latent hypermetropia is overcome in children by the action of accommodation, but it is not sustainable for a long duration when there are conditions of visual stress. Signs and symptoms like blurred vision, asthenopia, accommodative and binocular abnormalities, and squimt may develop. These features occur more commonly in manifest hypermetropia

Pathological hypermetropia is a rare type. It is greater in magnitude, and may be attributed to various causes: maldevelopment of the eye during prenatal or early postnatal period, corneal or lenticular changes, aphakia, chorioretinal or orbital inflammation or neoplasms,

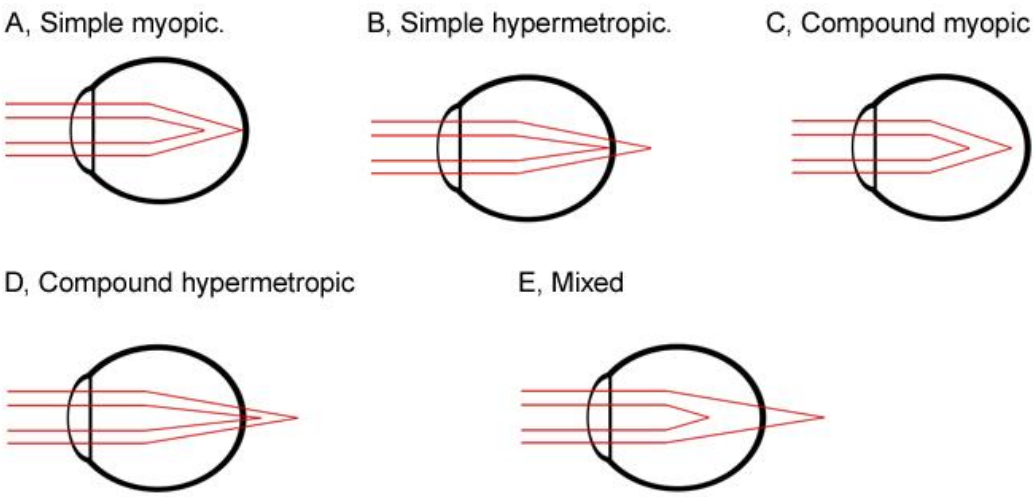
neurological or pharmacological causes, microphthalmia/nanophthalmia, anterior segment malformations etc. Numerous congenital and genetic developmental disorders and syndromes are also seen associated with high hypermetropia.

ASTIGMATISM

An optical system with astigmatism is one in which rays that propagate in two perpendicular planes have different foci.

Classification-

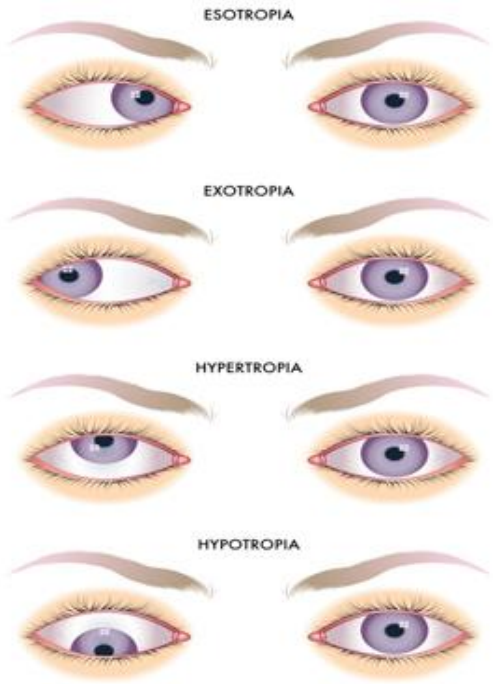
- Anatomical classification- Corneal astigmatism (Most common) and Lenticular astigmatism.
- Based on regularity of surface- Regular astigmatism and Irregular astigmatism
- Based on surface curvature- With-the-Rule- Vertical meridian of cornea more curved
    - Against-the-Rule-Horizontal meridian more curved.
- According to refraction-



STRABISMUS

Strabismus[23] or squint is the abnormal alignment of one or both eyes. It is one of the common manifestations in developmental delay.

It can be classified as follows:



HORIZONTAL

- **ESOTROPIA:** Abnormal convergence of visual axes (Inward deviation)

    - **Infantile esotropia:** Onset within first 6 months of life, usually with large and constant angle.
    - **Accommodative esotropia**: Onset between 18 and 24 months of age, often intermittent in the beginning, and usually associated with hyperopia (farsightedness, light focused behind the retina).
    - **Paralytic esotropia:** Palsy of abducent nerve (cranial nerve VI)

- **Restrictive esotropia:** Caused by an obstacle to the free movement of the eye (e.g., Graves' disease, orbital fractures)
- **Sensory esotropia:** Secondary to poor vision in one eye
- **Syndromic esotropia:** Associated with syndromes (e.g., Duane's retraction syndrome, Brown's syndrome).

- **EXOTROPIA:** Abnormal divergence of visual axes of the eyes (outward deviation)
  - **Infantile:** Onset within first 6 months of life, less common than infantile esotropia
  - **Idiopathic:** Intermittent or constant
  - **Paralytic:** Palsy of oculomotor nerve (cranial nerve III), may be associated with hypotropia, ptosis, and mydriasis (pupil dilation)
  - **Restrictive:** Caused by an obstacle to the free movement of the eye (e.g., orbital fractures and, less commonly than in esotropia, Graves' disease)
  - **Sensory:** Secondary to poor vision in one eye.

VERTICAL:

- **HYPERTROPIA:** It is the upwards deviation of the visual axis of one eye in relation to the other eye.
  - **Paralytic:** Palsy of trochlear nerve (cranial nerve IV)
  - **Restrictive:** Due to an obstacle to the free movement of the eye (e.g., Graves' disease, orbital fractures).
- **HYPOTROPIA:** It is the downward displacement of the visual axis of one eye relative to the other.

- **Paralytic:** Palsy of oculomotor nerve (cranial nerve III), may be associated with exotropia, ptosis, and mydriasis
- **Restrictive:** Caused by an obstacle to the free movement of the eye (e.g., Graves' disease, orbital fractures, Brown's Syndrome).

TORSIONAL (cyclotorsion):

- **INCYCLOTORSION:** Eye is rotated so that the superior pole is turned nasally and the inferior pole is turned temporally, thus the eye is 'rolled' toward the nose.
- **EXCYCLOTORSION:** Eye is rotated in such a way that the superior pole is turned temporally and the inferior pole is turned nasally, thus the eye is 'rolled' toward the ear.

FREQUENCY OF DEVIATION:

- **LATENT:** No evidence of strabismus while patient is fixating with both eyes.
- **INTERMITTENT:** Strabismus is present only intermittently, while at other times the eyes are aligned.
- **CONSTANT:** Strabismus is always present.

VARIATION WITH POSITION OF GAZE:

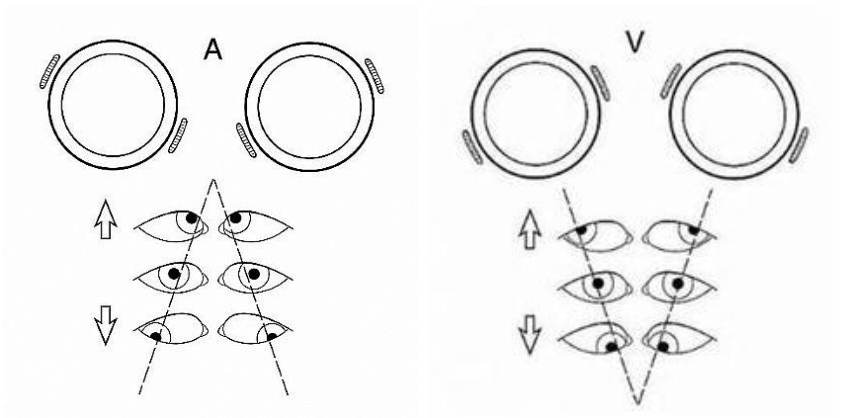
- **COMITANT (concomitant)-**

  Here the deviation of the eye do not vary in size with the gaze direction of the fixating eye. It is the most common form of childhood strabismus (except those associated with cranial nerve palsy or restriction).

- **INCOMITANT (non-comitant)-**

Deviation varies in size, with the direction of gaze of the fixating eye. Commonly paralytic or restrictive in nature and may point to underlying neurological or orbital disease. If horizontal strabismus alters in size between upgaze and downgaze, the pattern is described in comparison to letters.

- **A-pattern**: Increased exotropia (or decreased esotropia) in downgaze compared with upgaze. Considered clinically significant if difference is more than 10 prism dioptres.
- **V-pattern:** Increased esotropia (or decreased exotropia) in downgaze compared with upgaze. Considered clinically significant if difference is more than 15 prism dioptres.



- **X-, Y- and lambda-pattern** strabismus have also been described, but are more rare.

FIXATION:

- **Alternating-** Fixation spontaneously alternates from one eye to the other.
- **Monocular-** Here, definite preference exists for fixation with one eye over the other.

PAEDIATRIC CATARACTS

In the paediatric age group, cataract leads to more visual disability than any other form of treatable blindness. Children with untreated, visually significant cataracts, have their quality of life and socioeconomic status affected due to a lifetime of blindness. The blindness can be due to unoperated cataract, or complications of cataract surgery, or from ocular anomalies associated with cataracts. Other children suffer from partial cataracts that progress slowly over time, increasing the visual disability as the child grows. In children with developmental delay and bilateral cataract a careful history and examination will help in etiological diagnosis.

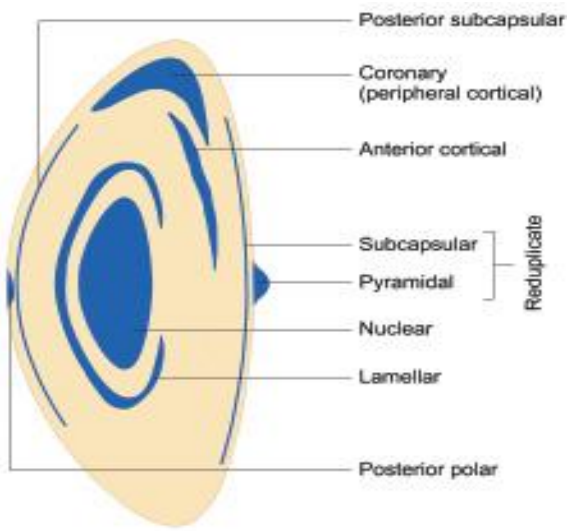
CLASSIFICATION

Cataracts in children can be classified using a number of methods including age of onset, etiology, morphology, genetic, secondary (Traumatic, inflammatory, iatrogenic)

1.AGE OF ONSET AND ETIOLOGY

Congenital/Infantile

The appearance of lens opacities at birth denotes a congenital onset. Yet the diagnosis of a lens opacity at a later stage does not rule out a congenital onset. Some structural categories of cataracts such as anterior polar, central fetal nuclear, and posterior polar clearly point to a congenital onset, whereas other types like cortical or lamellar may be associated either with a delayed onset or can be congenital in nature.



Acquired/Juvenile

Juvenile cataracts are those with an onset after infancy, regardless of underlying etiology.

Secondary

- Maternal infection (rubella)
- Uveitis
- Juvenile idiopathic arthritis
- Intraocular tumors
- Chronic retinal detachment

Iatrogenic

- Radiation
- Systemic steroids
- Vitrectomy
- Laser for retinopathy of prematurity

2.MORPHOLOGY

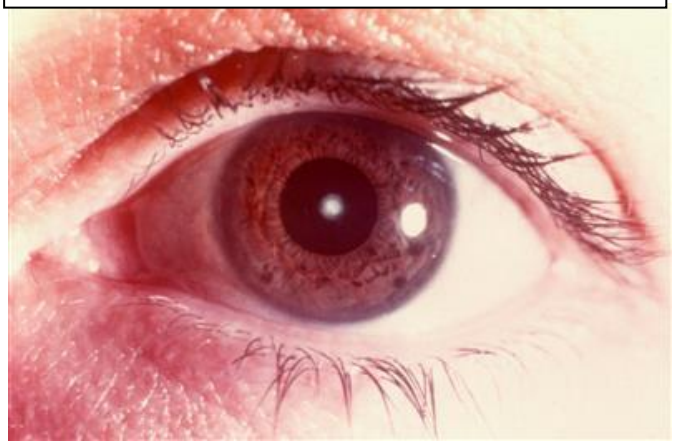
The morphology can throw light on the underlying etiology (isolated or associated with systemic disease), and probably on the visual prognosis following surgery.

Diffuse/Total- This is a common type of congenital cataract. These cataracts have no specific causes.

DIFFUSE/TOTAL

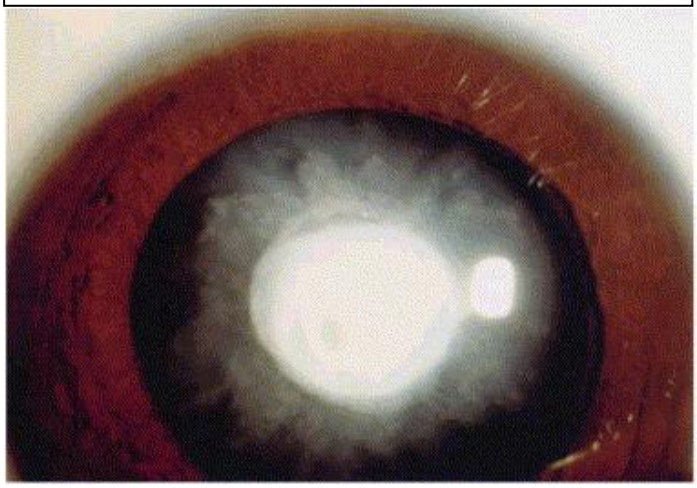
Anterior polar – The opacity is present in the capsule and can project into the anterior chamber as a small protruberence. There can be an underlying circular layer of cortical opacity a little larger than the polar opacity. These types of cataracts can be inherited dominantly, more often in bilateral cases. Unilateral cataracts are sometimes associated with anisometropia due to astigmatism or hypermetropia, which if left untreated can lead to amblyopia, even when the cataract is visually insignificant.

ANTERIOR POLAR



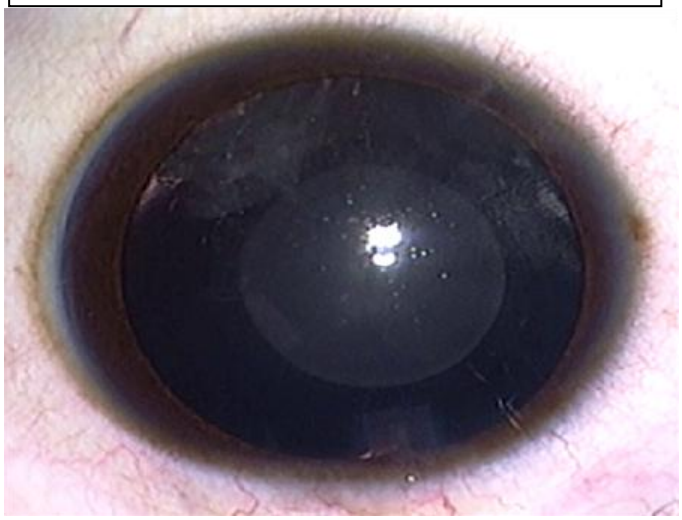
Pyramidal – They are generally larger than polar cataracts and tend to progress more to visual significance.

PYRAMIDAL



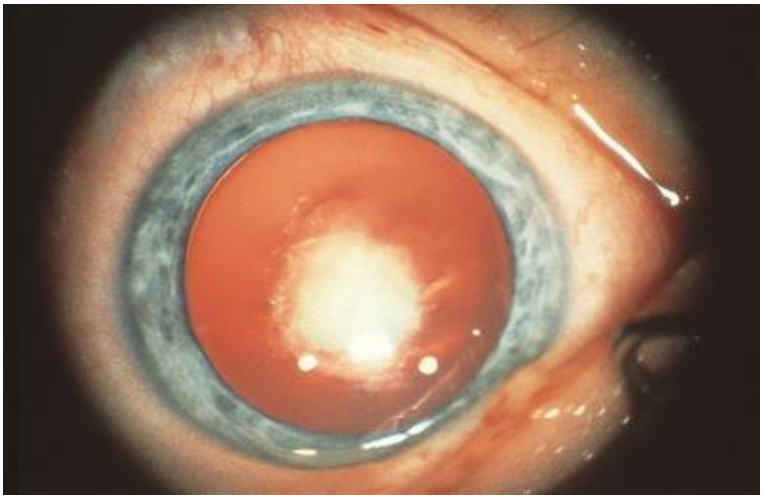
Cortical lamellar- In cortical lamellar cataract, the opacification is of a lamella, which is nothing but an ovoid layer of cortex, which is seen between adjacent clear lamellae. These often present with radial “rider” opacities. Familial lamellar cataracts are most often autosomal dominant in inheritance and have a good visual prognosis after removal.

CORTICAL LAMELLAR



Fetal nuclear-These opacities are seen in the most central part of the lens. They can occur as dot-like or dense opacities. These are usually 2-3.5 mm in size and can be associated with microphthalmia. There is a higher occurence of postoperative glaucoma in these cataracts due to associated microphthalmia.

FETAL NUCLEAR



Posterior polar-In these types of cataract, the opacity is in the posterior capsule itself. It is crucial to distinguish posterior polar from posterior subcapsular cataracts. These cataracts can be genetically determined and some of them are seen with mutations in *PITX3*

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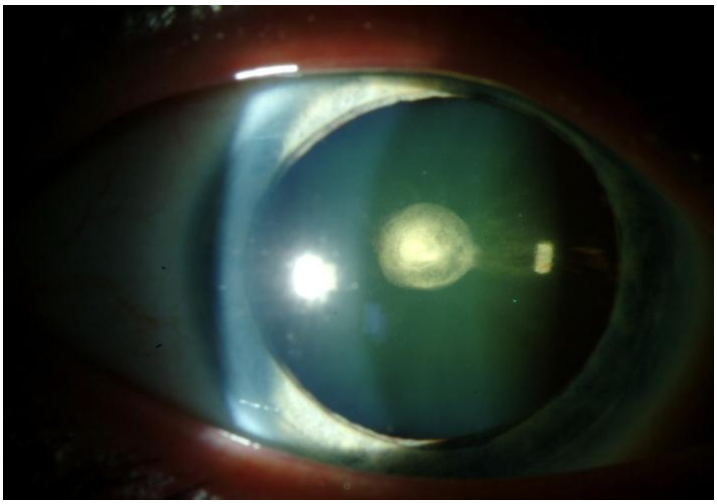
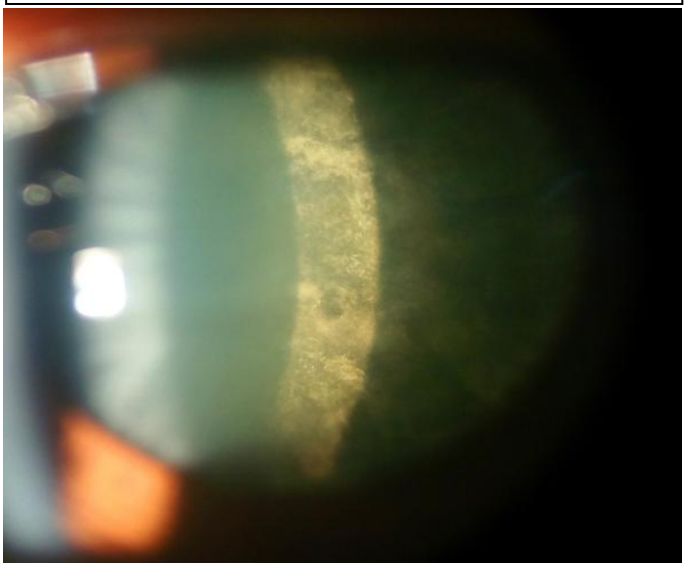
POSTERIOR POLAR



Posterior subcapsular-These cataracts can be congenital but are more often acquired as a consequence of trauma or steroid usage. The opacities are in the cortex with no involvement of the capsule proper.

POSTERIOR SUBCAPSULAR



CORTICAL VISUAL IMPAIRMENT

Cortical Visual Impairment (CVI)[5] is considered a temporary or permanent form of visual defect due to a disturbance of the posterior visual pathways with or without involvement of the occipital lobes of the brain. It is a 'spectrum' condition, in which all the features do not manifest in every child or in every age group.

The amount of vision impairment can extend from severe visual impairment to total blindness. The degree of neurological damage and visual deficit is dependent on the age of onset and the location and degree of the insult. It is a condition that denotes that the cerebral visual systems do not constantly perceive or interpret what the eyes see. The presence of CVI does not indicate the child's cognitive ability.

The definition includes 3 criteria-

- Normal ocular examination
- Decreased visual acuity
- Confirmation of damage to the cerebral or posterior visual paths.

A typical feature of CVI is improvement in visual function. This may also be attributed to a very slowly maturing visual system.

CAUSES- The main causes of CVI include:

- Asphyxia
- Hypoxic ischemic encephalopathy (HIE)
- Periventricular leukomalacia (PVL)
- Developmental brain defects
- Head injury
- Hydrocephalus
- Neonatal hypoglycemia
- Infections of the central nervous system, such as meningitis, and encephalitis.
- Antenatal drug use by the mother

FEATURES

In the initial stages, children with CVI seem blind, with vision improving later. The child should be examined by both paediatric neurologist and paediatric ophthalmologist.

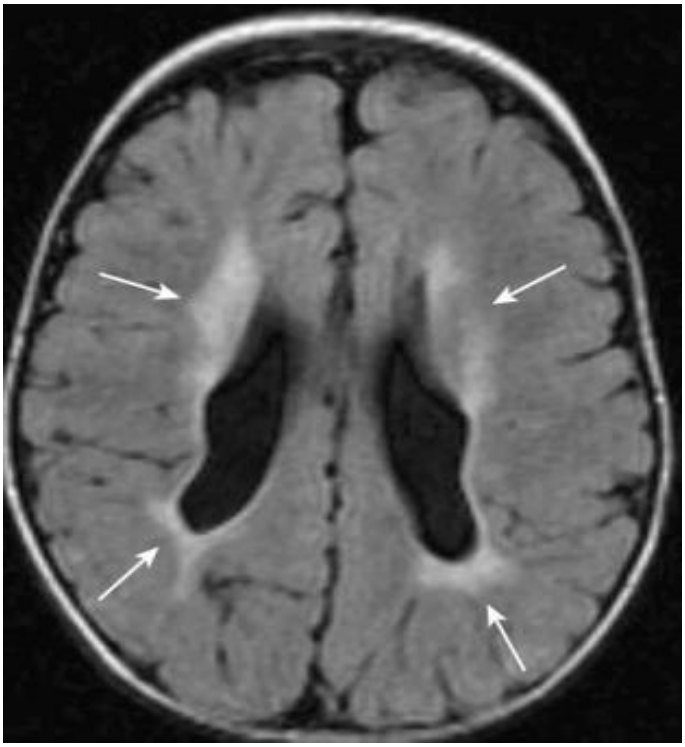
It is a difficult diagnosis to make.CVI is diagnosed when:

- Child has poor or no visual response

- Normal pupillary reactions with a normal ophthalmic examination.
- Extraocular movements- most often normal.
- Visual functioning-variable.

The report of an **MRI** (Magnetic Resonance Imaging) in accordance with an examination, make up the basis for diagnosis. Findings in MRI include changes of periventricular leukomalacia such as abnormal dilatation and irregularity of the lateral ventricles, high intensity signals in the periventricular white matter on T2 weighted images and periventricular gliosis.

MRI Brain with PVL



Behavioural/Visual Features

The occurrence of and type of added handicaps usually vary. Some children have adequate language skills whereas others do not. Spatial confusion is commonly seen in children with CVI due to the proximity of the occipital and parietal lobes of the brain.

Symptoms:

The most common CVI symptoms are:

- Abnormal light response - light gazing OR photophobia (seen in one third of children)
- Blunted or avoidant social gaze
- Brief fixations with intermittent following
- Use of peripheral vision more productively than central vision.
- Poor visual acuity
- Visual field loss - generalized constriction, inferior altitudinal, hemianopic defect
- Colour vision is usually retained in children with CVI (colour perception is depicted bilaterally in the brain, and is less vulnerable to complete elimination).
- Children may show poor depth perception, which influences their capacity to reach for a target.
- Vision can be better when either the visual target or the child is moving.

OCULAR EXAMINATION: The ophthalmic examination may show-

- Anomaly of the optic nerves –Pallor, large cup (that is not severe enough to cause visual impairment present in the child)
- Strabismus-common
- Nystagmus-less common.
- Pupillary reactions are usually normal.
- Refractive error correction may enhance some visual behaviour and should be attempted if present.
- Close viewing is practised by the child, to magnify the object or to decrease crowding.

OPTIC ATROPHY

Optic nerve atrophy is not a disease entity, but a sign that warns the ophthalmologist of a possibly more serious condition. A complete evaluation of a child presenting with optic atrophy is, thus, mandatory, and a complete understanding of the prevailing causes helps the physician in an suitable work-up and subsequent management.

This is specifically important where prompt diagnosis and intervention of a progressive or life-threatening cause may reduce the visual defect and general morbidity in the forth-coming years. It also guides the family towards the needed visual, educational and social rehabilitation.

It is the final common morphological endpoint of any disease entity that leads to axon degeneration in the retinogeniculate pathway. Optic atrophy manifests as alterationss in the colour and morphology of the optic disc which is associated with variable levels of visual dysfunction.

The Kestenbaum index is defined as the number of capillaries seen on the optic disc. The normal count is around 10. When the number of capillaries on the disc goes down to 6 or less, it denotes optic atrophy.If the number is more than 12, it indicates disc hyperemia.

CLASSIFICATION

Optic atrophy is classified on 3 basis- pathological, ophthalmoscopic and etiologic.

1.Pathological:

- **Anterograde degeneration (Wallerian degeneration)-** The degeneration starts in the retina and progresses towards the lateral geniculate body, as seen in toxic retinopathy, chronic simple glaucoma etc.
- **Retrograde degeneration-** The degeneration begins from the proximal part of the axon and progresses towards the optic disc, as seen in optic nerve compression via intracranial tumor.
- **Trans-synaptic degeneration-**In this type of degeneration, the loss of neuron on one side of a synapse causes a degeneration of the neuron on the other side, as seen in individuals with occipital damage in utero or during early infancy.

2.Ophthalmoscopic optic atrophy:

- **Primary optic atrophy-** In primary atrophy, usually caused by pituitary tumour, optic nerve tumour, traumatic optic neuropathy, multiple sclerosis, the optic nerve fibers tend to degenerate in an orderly fashion and are then replaced by glial cell columns with no alteration in the optic nerve head architecture.

  The colour of the disc is chalky white and margins are well demarcated, with normal retinal vessels.

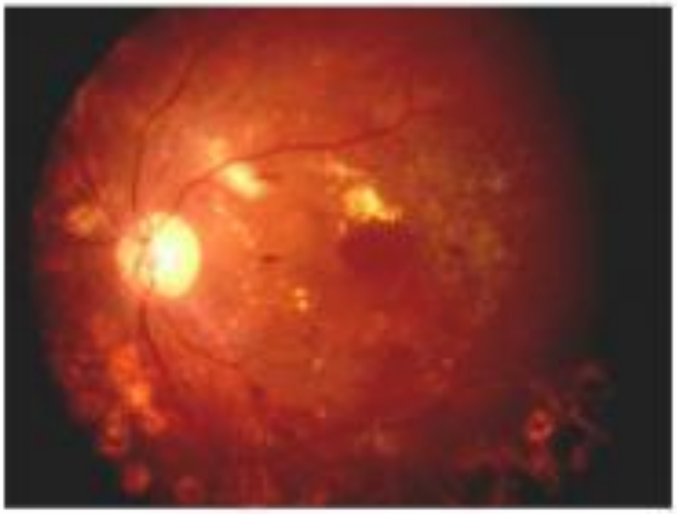
PRIMARY OPTIC ATROPHY

- **Secondary optic atrophy**-In cases with secondary optic atrophy which usually follow papilledema or papillitis, the atrophy is a result of the edema. The disc is generally dirty grey in colour, with poorly defined margins, with the lamina cribrosa being obscured due to proliferating fibroglial tissue.

SECONDARY OPTIC ATROPHY

- **Consecutive optic atrophy-**Here, the usual causes include retinitis pigmentosa, myopia or central retinal artery occlusion. The disc has waxy palour with distinct disc margin, marked arteriolar attenuation, and a normal cup.

CONSECUTIVE OPTIC ATROPHY



- **Glaucomatous optic atrophy-**It is also known as "cavernous optic atrophy", characterised by marked cupping of the disc.

3.Etiologic optic atrophy:

- **Hereditary atrophy**[4]**-**This type of atrophy is divided into congenital or infantile (which may be recessive or dominant), Behr hereditary optic atrophy- autosomal recessive, and Leber optic atrophy.

    - Autosomal dominant optic atrophy **Type 1** is due to mutations in the *OPA1* gene on chromosome 3q29.
    - X-linked optic atrophy **Type 2** is due to mutation in the *OPA2* gene on Xp11.4-p11.21. The child presents with early-onset vision loss which is slowly progressive.

- Hereditary optic atrophy **Type 3** is due to mutation in the *OPA3* gene on 19q13.32. This mutation is associated with early-onset visual loss with cataract.

- **Other Types-**Consecutive atrophy ,circulatory atrophy (vascular) ,metabolic atrophy, demyelinating atrophy, pressure or traction atrophy, postinflammatory atrophy, traumatic optic neuropathy.

OPTIC ATROPHY IN CHILDREN

Hereditary and congenital types of atrophy usually manifest in the first two decades of life. They are classified into the 3 major groups:

- Optic atrophy associated with generalized white matter lesions like adrenoleukodystrophy.
- Optic atrophy with unrelated systemic features (seen with OPA1 gene mutation)
- "Isolated optic atrophy"-It can be autosomal dominant or recessive with mitochondrial inheritance like Leber hereditary optic neuropathy.

NYSTAGMUS

Nystagmus is defined as a periodic rhythmic oscillation of the eyes. The oscillations can be sinusoidal and of almost equal amplitude and velocity (pendular nystagmus) or, more often, with a slow initial phase and a rapid corrective phase (jerk nystagmus).

Nystagmus[6] can be-

- Unilateral or bilateral, but, unilateral nystagmus only appears unilateral, but is actually due to asymmetry between the two eyes.
- Conjugate or disconjugate (dissociated).
- Horizontal, vertical, torsional (rotary), or any combination of these movements superimposed upon each other.
- Congenital (associated with afferent visual pathway abnormalities-sensory nystagmus) or acquired (commonly caused by abnormalities of vestibular input.

MECHANISM- Foveal centration of a target of concern is needed to acquire the highest level of visual acuity. Three mechanisms are involved in maintaining foveal centration of an object of interest: fixation, the vestibulo-ocular reflex, and the neural integrator. Nystagmus occurs when any of these mechanisms fail. Thus, nystagmus reflects a failure of early sensorimotor integration (Especially in congenital nystagmus).

CONGENITAL NYSTAGMUS (Infantile nystagmus syndrome)

It is recognized in the first few months of life affecting both sexes equally. It can be associated with a positive family history. These children do not have oscillopsia and the nystagmus is abolished during sleep.

Congenital nystagmus may present with poor vision or good acuity. The ophthalmologist should rule out any damage to the visual pathways. More often, electrophysiologic tests (ERG, VEP) are needed. 15% of the cases present with strabismus.

Congenital nystagmus often occurs with conditions such as ocular albinism, achromatopsia, Leber congenital amaurosis, and aniridia.

It is divided into-

- **Afferent (sensory deficit) nystagmus**-More common; due to visual impairment.
- **Efferent (idiopathic infantile) nystagmus**, which is due to oculomotor abnormality.

Newer additional subtypes of infantile nystagmus include-

(1) Nystagmus associated with albinism

(2) Latent and manifest latent nystagmus

(3) Spasmus nutans-Occurs in otherwise healthy children and resolves within one year of onset.

ACQUIRED NYSTAGMUS

Both focal and diffuse disorders affecting any of the 3 mechanisms that control eye movements may cause acquired nystagmus.

Causes

- **Seesaw nystagmus** is caused by rostral midbrain lesions, lesions of the parasellar region (eg, pituitary tumors) and secondary to retinitis pigmentosa.
- **Downbeat nystagmus** is caused by vestibulocerebellar lesions and lesions in the medulla (eg, Arnold-Chiari malformation, demyelination) and also heat stroke. Around 50% of the cases do not have an identifiable cause.
- **Upbeat nystagmus** is caused by lesions of the anterior vermis of the cerebellum , medulla and benign paroxysmal positional vertigo.
- **Periodic alternating nystagmus** is due to ocular malformations, demyelination, trauma, ocular media opacities and infections.
- **Pendular nystagmus** is usually seen in demyelinating disease,monocular or binocular visual deprivation, brain stem or cerebellar dysfunction.
- **Torsional** - Lateral medullary syndrome (Wallenberg syndrome)
- **Abducting nystagmus** of internuclear ophthalmoplegia
- **Gaze evoked**-Due to drugs like anticonvulsants (eg, phenobarbital, phenytoin, carbamazepine) and alcohol.

VISUAL ACUITY ASSESSMENT

1. Tests for indirect assessment of vision-

a) History and observational tests

b) Binocular fixation preference and fixation targets

c) CSM method

2. Tests for recognition acuity-

a) Dot visual acuity

b) Coin test

c) Miniature toy test

d) Marble game test

e) Bock's candy test (100's and 1000's test)

f) Worth ivory ball test

g) Kay pictures and LEA symbol tests

h) Sheridan Gardiner test

3) Tests for resolution acuity-

a) Opticokinetic nystagmus

b) Preferential looking tests

c) Visual evoked potentials

d) Landolt C test/Snellen Charts

1.TESTS FOR INDIRECT ASSESSMENT OF VISION-

History and observation:

- Parents/guardians are routinely questioned whether their child reacts to smile, enjoys silent mobile phones or follows objects.
- Observations by physicians include strabismus, nystagmus, persistent staring gaze, and inattention to objects.

Binocular fixation preference and fixation targets:

- With ideal targets, this reflex can be elicited by 6 weeks of age.
- The object of visual interest, most commonly a coloured toy, is moved from side to side. The physician observes the child's eyes turning toward the object and whether the child follows its movements (fix and follow behaviour). The examiner may occlude one of the infant's eyes to test each eye separately.
- If the infant has fix and follow behaviour, then it is presumed that he/she can visualize a small target or toy in an ideally lit room.
- The face is a better target than test objects. Thus, if following movement is not demonstrated with test object, it should be repeated with the face of the mother/guardian used as test stimulus.

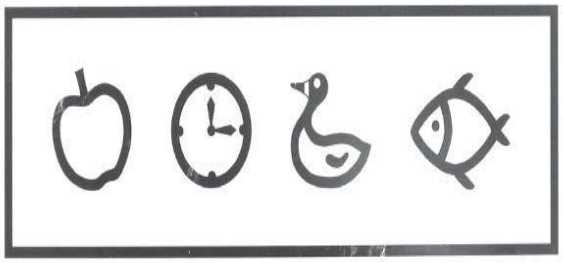
CSM METHOD-

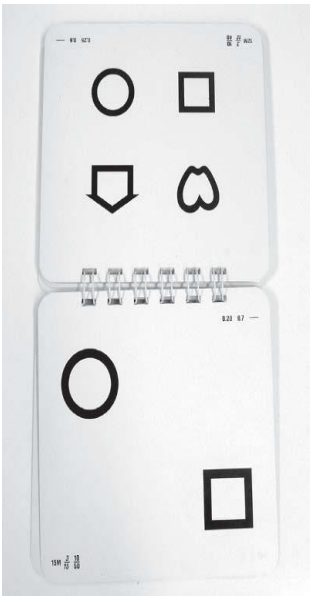
- It is tested with one eye fixing on a target kept at 40 cm, with accommodation in place.
- **'C' is for "central"**-It is the location of corneal reflex when the patient fixes on the examiner's light under uniocular conditions. Under normal conditions, light reflected from the cornea falls at the centre of the cornea and it is found symmetrically in both eyes. If fixation target is seen eccentrically, the fixation is called uncentral.
- **'S' is "steadiness"** of fixation on the testing light when it is kept steady without movement and also when it is slowly moved.
- **'M' is "Maintained"-** It is the ability of the child to maintain the alignment initially with one eye, and later with the other, when the other eye in uncovered. Maintenance of fixation is also examined under binocular conditions. Failure to maintain fixation with each eye, with opposite eye uncovered is presumed to be evidence of a disparity in visual acuity between the two eyes.

- **CSM=Central Steady Maintained→ 6/6 – 6/9**
- **CSUM=Central Steady Unmaintained→ 6/36 – 6/60**
- **CUSUM=Central Unsteady Unmaintained → <6/60**

2.TESTS FOR RECOGNITION ACUITY:

- **DOT VISUAL ACUITY TEST**: An illuminated box with different sized black dots printed on it is shown to the child. The smallest dot seen represents the visual acuity of the child.
- **COIN TEST**: Child is made to identify two faces of coins of varying sizes shown from different distance.
- **MINIATURE TOY TEST:** Child is made to see a miniature toy from 10 feet distance and asked to name or pick the pair from the lot.
- **MARBLE GAME TEST**: The child is asked to place marbles in holes of a card or box. It compares the vision of one eye when the other is closed and it is recorded as useful or less useful.
- **WORTH IVORY BALL TEST:** Ivory balls 0.5 to 2.5 inches in size are rolled on the floor and the child is asked to pick up each. Acuity is evaluated according to the smallest size for the test distance.
- **BOCK'S CANDY BEAD TEST:** Snellen equivalent of 6/60 is tested by this method. The child is asked to match 1 mm size pick up beads from 40 cm.
- **KAY PICTURES TEST** (done at 6 meters) and **LEA SYMBOLS TEST** (done at 3 meters)



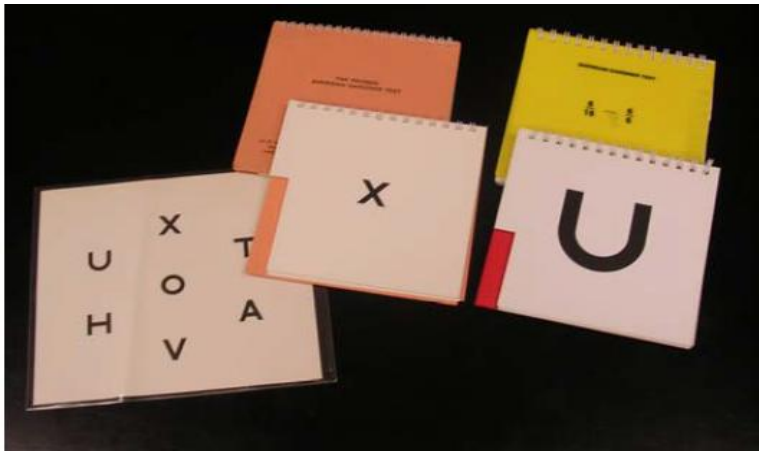


KAY PICTURES **LEA SYMBOLS**

- **SHERIDAN GARDINER TEST –**

  It is used for children in the ages of 2-5yrs . The test is done at a distance of 6 meters and if visual acuity is less than 6/60, it can be done at 3 meters. The chart is made in a booklet form, consisting of letters HOTV in different sizes , arranged in circular form to provide proper orientation.

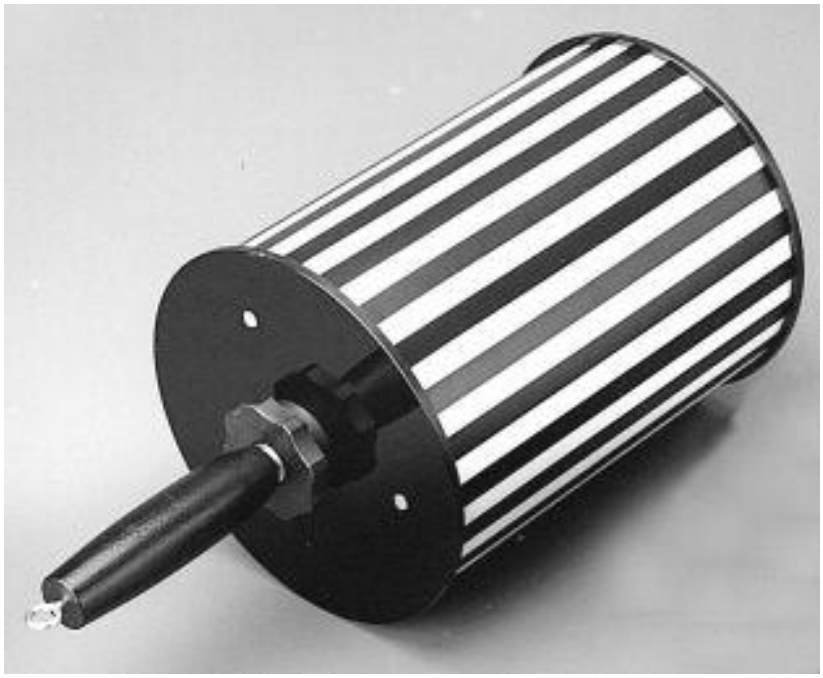
  'HOTV' letters are specifically used because they are considered the standard letters for visual acuity measurement. A key card is given and the child is asked to match it with the letter in the main card.



3.TESTS FOR RESOLUTION ACUITY:

OPTICOKINETIC NYSTAGMUS-

- Evaluation of the presence or absence of opticokinetic nystagmus was the first machine assisted approach to visual acuity assessment in preverbal children.
- Binocular acuity is tested when the child is positioned on his or her back looking up at black and white stripes. The stripes are moved in an arc around 180^{o} of the child's visual field.
- Child follows the stripe with a steady motion and when it disappears suddenly, he/she picks up a new stripe.
- A measurement of visual acuity is made by changing the width of stripes or the distance from the drum.

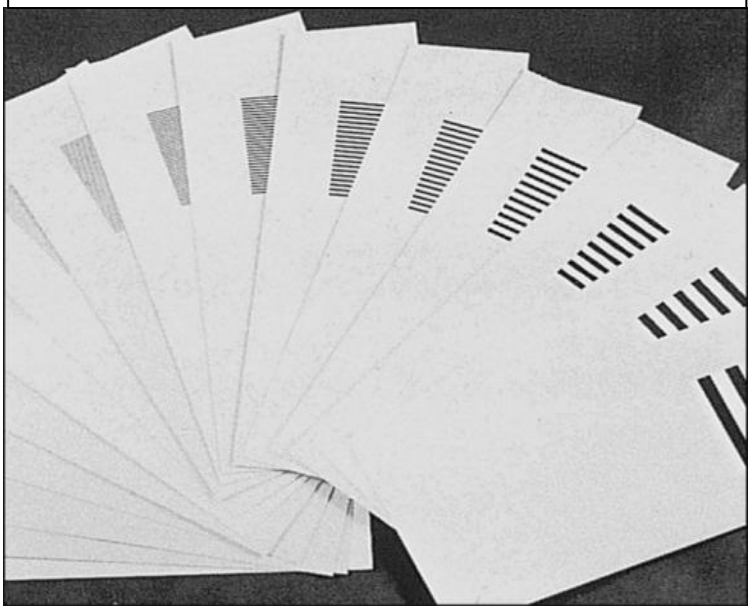


FORCED CHOICE PREFERENTIAL LOOKING:

- This technique was started by David Teller.
- It is based on the fact that infants display a greater tendency to fix a pattern stimulus better than a homogeneous field.
- This technique measures resolution acuity, with the help of either :

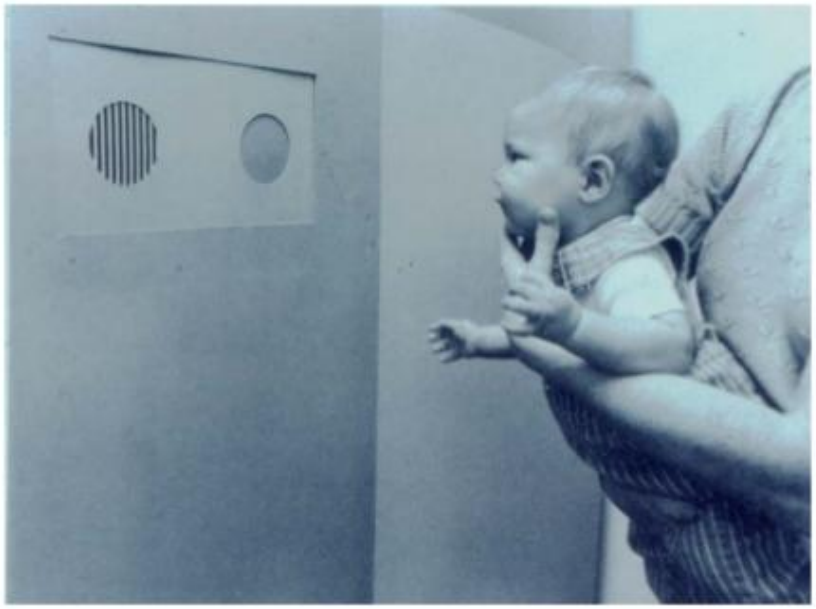
1. A grating target-Teller cards[8]

TELLER ACUITY CARDS



2. The “vanishing optotype principle”-Cardiff Acuity Cards.

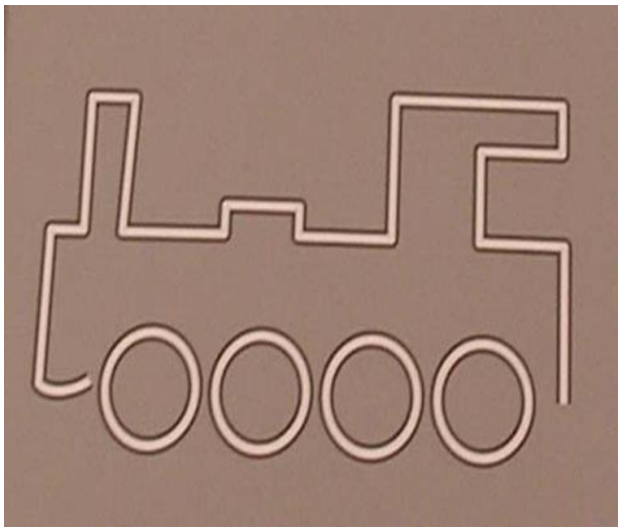
- Preferential looking requires showing the child two stimuli, a grating made up of black and white stripes (or other quantified patterns), and a grey screen with equal "space-average" luminance.



- The observer, not aware of the location of the stimuli, stands behind a peephole located centrally between the grating and the homogeneous field.
- The observer monitors the direction of movement of the infant's eyes and head at the time of stimulus presentation. The width and position of the stripes are changed every trial.
- Acuity is measured by assessing the smallest striped width in which the child shows differential fixation of the grating in contrast to the homogeneous field. Thus the frequency of the line spacing measures the visual acuity.

THE CARDIFF TEST:

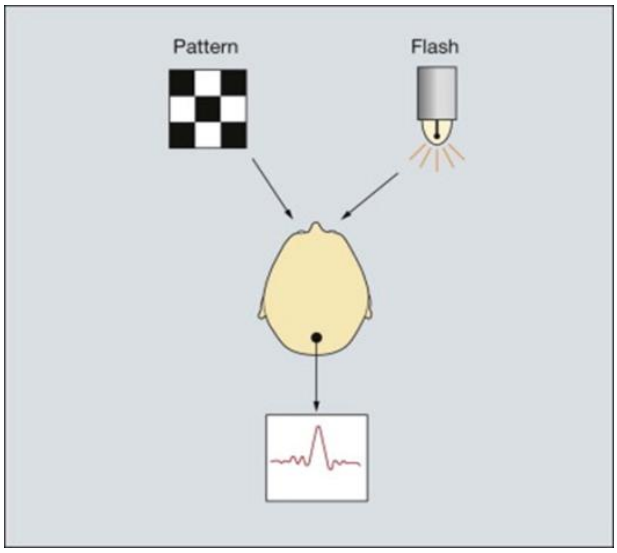
- This test measures both resolution acuity and recognition acuity. It is an appropriate test for children between 18 - 60 months. It consists of different cards, that are held in front of the child. Every card has one picture in the upper or the lower part. As the child looks in the direction of the picture on the card, the size of the picture as seen, is noted.
- The targets are pictures drawn with a white outline enclosed in between two black bands, on a grey background. The overall brightness of the picture is same as that of the grey background.
- When the child is able to resolve the two different bands, the picture becomes visible, thus confirming good visual acuity, but if the bands are too thin for the child to resolve, the picture merges with the grey background, thus becoming invisible. These are called “Vanishing optotypes”.



VISUAL EVOKED POTENTIALS:

- Visual evoked potentials (VEPs) are electrical brain responses that are triggered by the presentation of a visual stimulus.
- VEPs are differentiated from the spontaneous electroencephalogram (EEG) by their consistent time of occurrence after the presentation of the stimulus, termed as “time-locking”.
- **Types :**

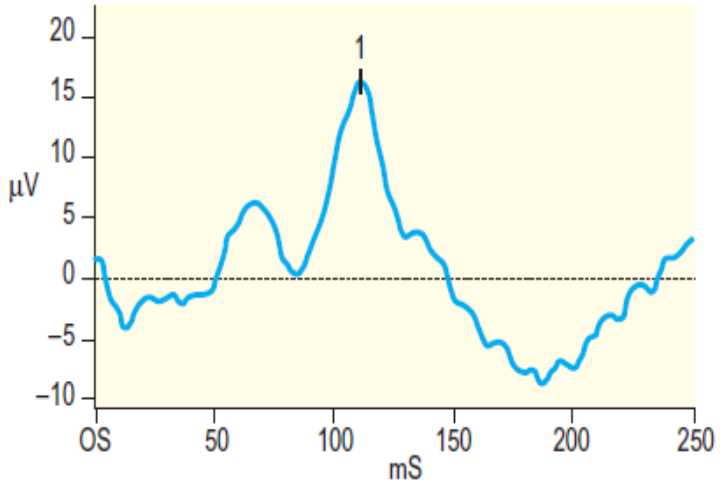
1. Flash VEPs
2. Pattern reversal VEPs
3. Sweep VEPs



1.Flash VEP

- The response to simple flashes of light is useful where the visual acuity is too poor to perceive even large checks or where fixation/concentration is poor.

- The flash VEP is not dominated by the macula as the pattern VEP and can thus it can be recorded through media opacities.
- Unfortunately, the appearance of flash VEP is more variable than the pattern VEP and, generally, it is used to provide a basic indication of the integrity of the visual pathway from the eye to the occipital cortex.
- The most consistent feature is a positive component-P2, occurring at around 125 ms after the flash, but identification of even this component is difficult.



2.Pattern VEP

- Pattern VEP is used to measure visual acuity by estimating the response amplitude of the VEP at each frequency, for a given range of spatial frequencies.
- Pattern reversal VEP is measured using a checker board pattern.
- The pattern of stimulus is changed (black squares→ white and white squares→ black) with the overall illumination remaining same.
- It depends on form sense and thus gives a rough estimate of visual acuity.

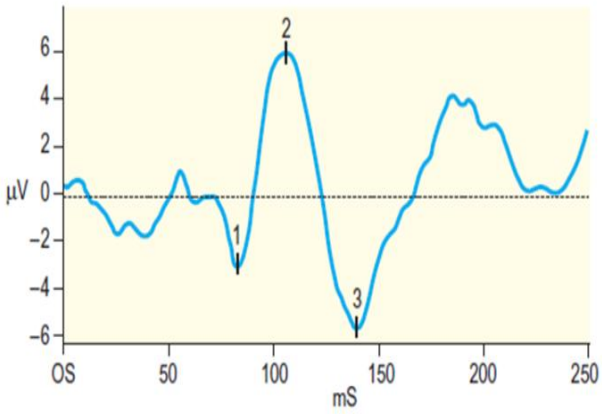
- When the size of the checks is decreased to a level where the contrast borders cannot be resolved, the cortical response disappears.

- Visual acuity of approximately-

    - 6/18–6/24 is required for a clear cortical response when the check size subtends a visual angle of 15 min arc.
    - 3/60–6/60 is required for a clear cortical response at a check size of 60 min arc.

- The most important component is the P100 response, which is a positive peak with a latency of approximately 100 ms . The latency of P100, and symmetry over the two sides of the scalp are noted.

- Amplitude is also be measured, but changes in size are less helpful for the detection of pathology.



Normal pattern visually evoked potential to 60 min arc reversing checkerboard stimulus. The key features are: (1) N75; (2) P100; and (3) N135.

3.Sweep VEP :

- Sweep VEP essentially performs the same function, but the spatial frequencies are varied from low to high in about 10-20 seconds rapidly and amplitudes are plotted instantly with respect to spatial frequency (or time).

ACUITY MEASUREMENT ACCORDING TO AGE-

Age	Suitable Visual Acuity Test
<18mths	Response to occlusion/CSM Method Bock candy beads (100's and 1000's) Keeler acuity cards (Forced-choice preferential looking test) Stycar graded balls test Cardiff acuity cards
18mths - 3 years	Keeler acuity cards Cardiff acuity cards Kay picture test Sheridan-Gardiner test Sonksen-Silver test
3-5 years	Kay picture test Sheridan-Gardiner test Sonksen- Silver test Cambridge crowding cards Glasgow acuity cards Stycar test
5 + years	Sheridan-Gardiner test Sonksen-Silver test Cambridge crowding cards Glasgow acuity cards Stycar test Maclure test (near) Snellen or LogMAR test

PART-II

AIM OF THE STUDY

This study was done to analyze the various causes of visual morbidity in children with delayed milestones in the age range of 6 months to 5 years, with the aim of improving early detection of ocular abnormalities in these children.

The following were the 3 primary aims:

- To study the various ocular manifestations in children with delayed milestones.
- To study antenatal, natal and postnatal factors present in these children with ocular manifestations.
- To study the types of developmental delay present in the children with ocular manifestation, whether isolated or global delay.

INCLUSION CRITERIA

Children aged 6 months to 5 years, diagnosed with any type of developmental delay (global or otherwise), attending the ophthalmology outpatient department (referred from paediatric neurology OPD) were included in the study.

EXCLUSION CRITERIA

- Children with neuromuscular disorders causing motor abnormalities were excluded.
- Children with associated syndromes were excluded from the study.

MATERIALS AND METHODS

This study was performed in children aged 6 months to 5 years, with delayed developmental milestones, who attended the Ophthalmology out-patient department (referred from Paediatric Neurology OPD), Govt Stanley Medical College and Hospital.

After prior consent from the parent/guardian, a detailed history regarding:

- Antenatal period
- Natal period
- Postnatal period, were obtained from the parent/guardian.

History regarding attainment of developmental milestones in all four domains (gross motor, fine motor, language, social) was elicited.

A preliminary general examination of the child was done.

Detailed ophthalmic examination was done, consisting of:

- Visual acuity assessment-Methods of acuity assessment varied according to age.
    - Children up to 3 years- Central Steady Maintained (CSM) method was used, for the purpose of standardisation, as other methods were not reliable in some of these children with cognitive and language delays.
    - Children above 3 years- Sheridan Gardiner HOTV test was employed.

- Anterior segment examination- including slit lamp examination (when possible), squint assessment, nystagmus evaluation.

- Dilated fundus examination (both direct and indirect).

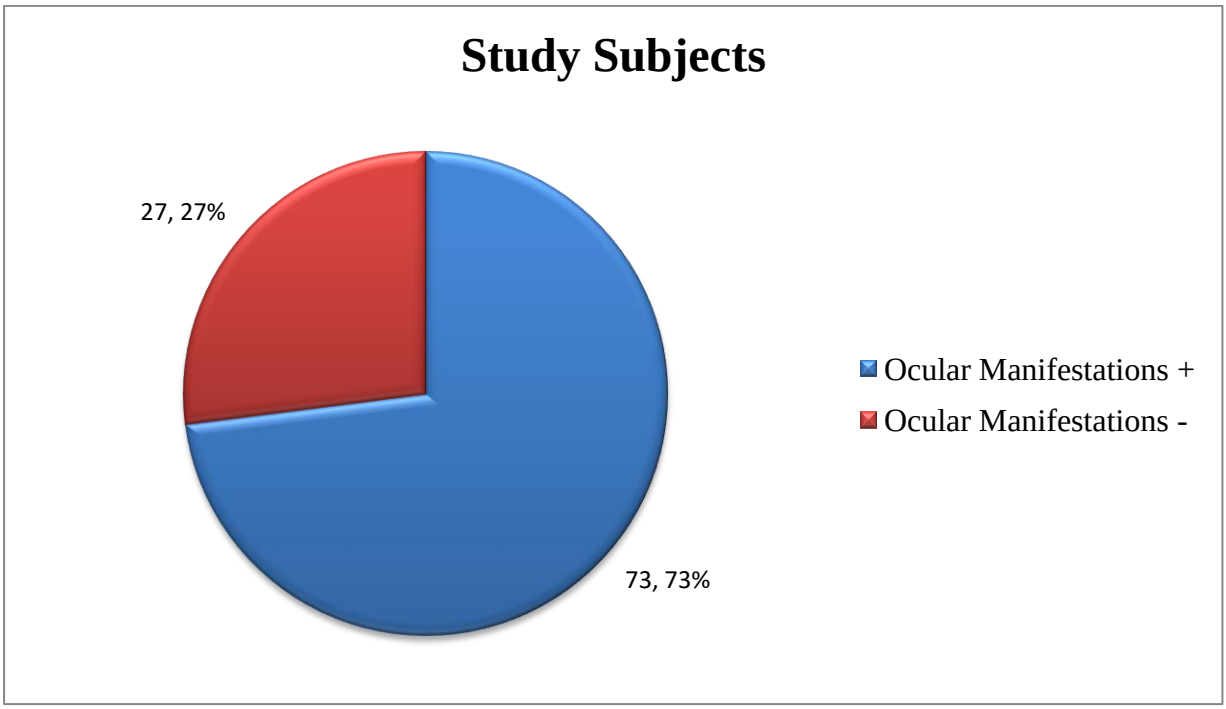
- Retinoscopy using streak retinoscope.

- Subjective refraction was done whenever possible.

OBSERVATIONS AND RESULTS

STUDY SUBJECTS

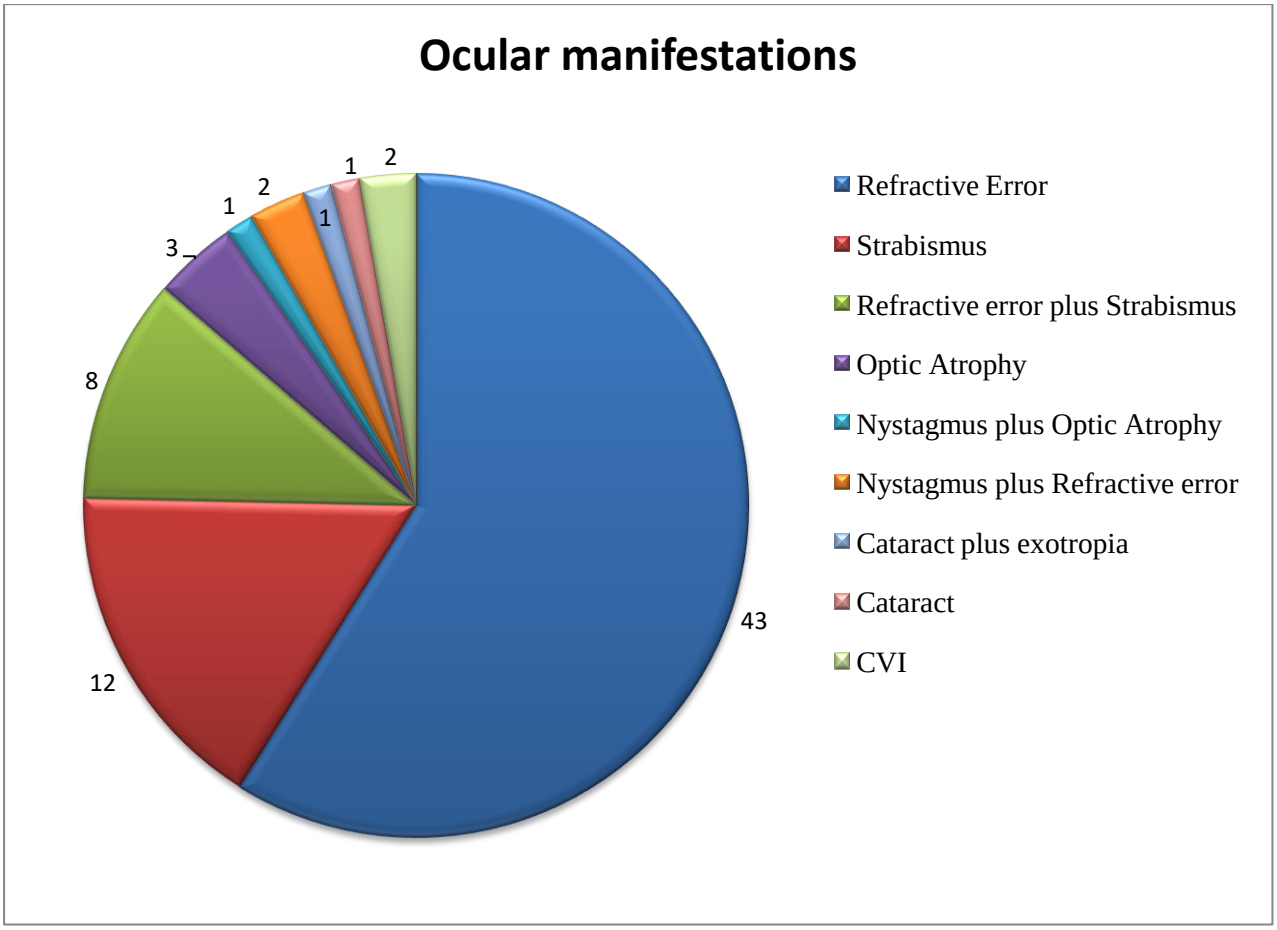
- The total number of children with ocular manifestations among the 100 was 73.



Study Subjects	Number	%
Ocular Manifestations +	73	73.00
Ocular Manifestations -	27	27.00
Total	100	100.00

OCULAR MANIFESTATIONS

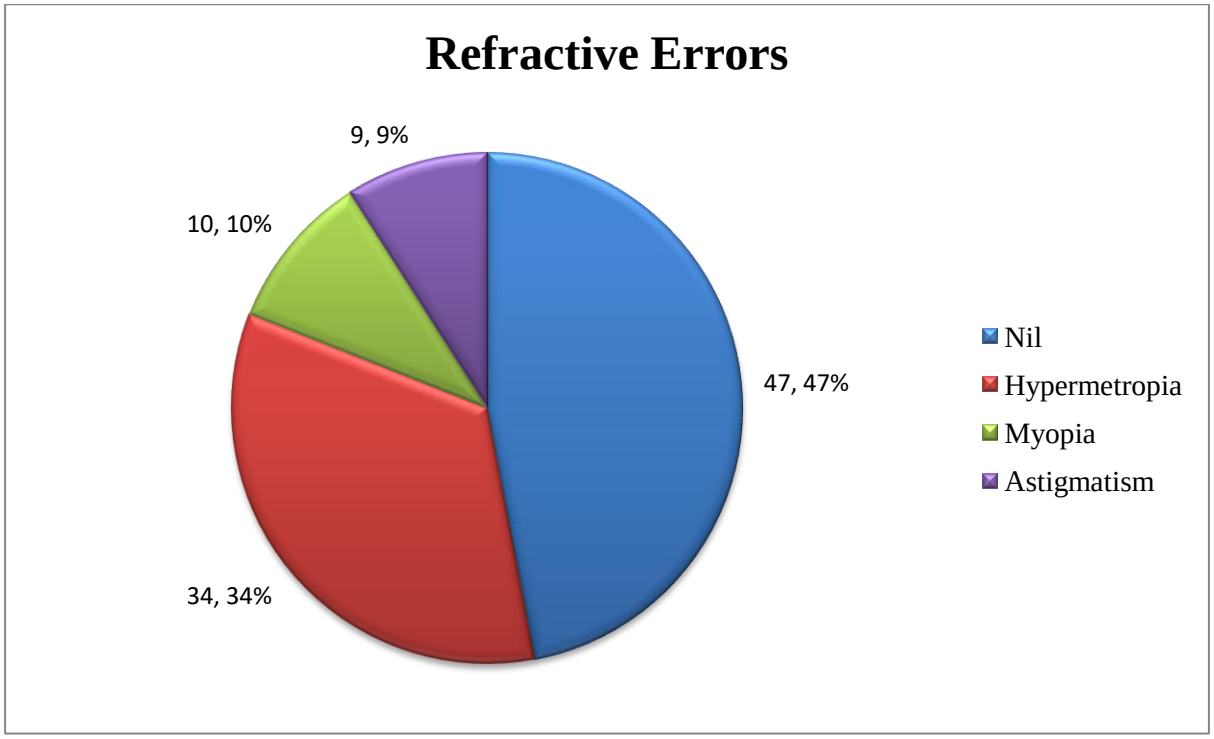
- Among the manifestations, the most common was found to be **refractive errors**, with a prevalence of 53 cases.
- The second most common manifestation was **strabismus** (21 cases)
- Other ocular manifestation seen were- Optic atrophy (4 cases), Cataract (2 cases), Nystagmus (3 cases) and cortical visual impairment (2 cases).
- Among these,there were 8 cases with refractive error and strabismus, 1 case with nystagmus and optic atrophy and two cases with nystagmus and refractive error.



MANIFESTATION	NUMBER	PERCENTAGE (%)
Refractive Error	43	58.90
Strabismus	12	16.43
Refractive error plus Strabismus	8	10.95
Optic Atrophy	3	4.10
Nystagmus plus Optic Atrophy	1	1.36
Nystagmus plus Refractive error	2	2.73
Cataract plus exotropia	1	1.36
Cataract	1	1.36
CVI	2	2.73
TOTAL	73	100

REFRACTIVE ERRORS

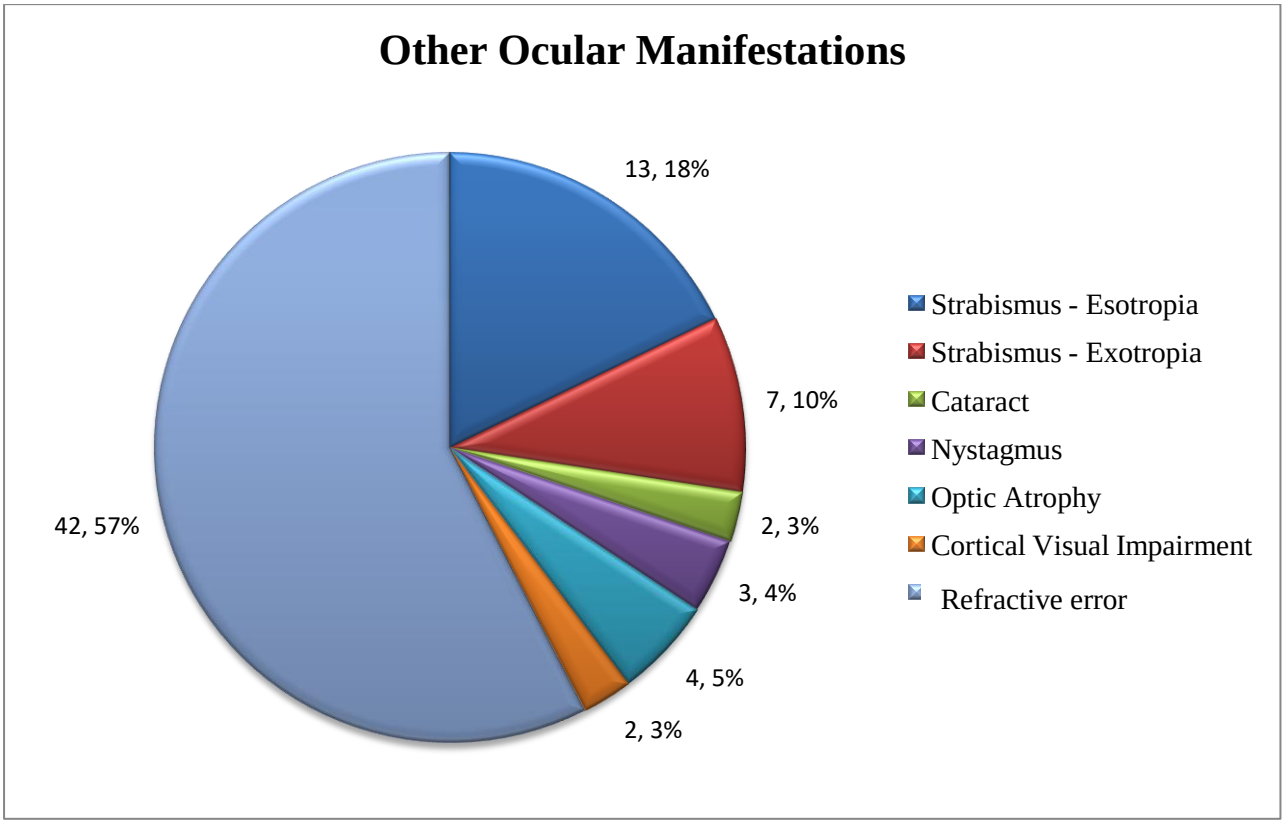
- Out of the 53 cases of refractive errors, the most common error was **hypermetropia** (**34 cases**). 10 cases had myopia and 9 had astigmatism.
- Among astigmatism-5 were compound myopic, 3 were compound hypermetropic and 1 was mixed astigmatism.



Refractive Errors	Number	%
Nil	47	47.00
Hypermetropia	34	34.00
Myopia	10	10.00
Astigmatism	9	9.00
Total	100	100.00

OTHER OCULAR MANIFESTATIONS

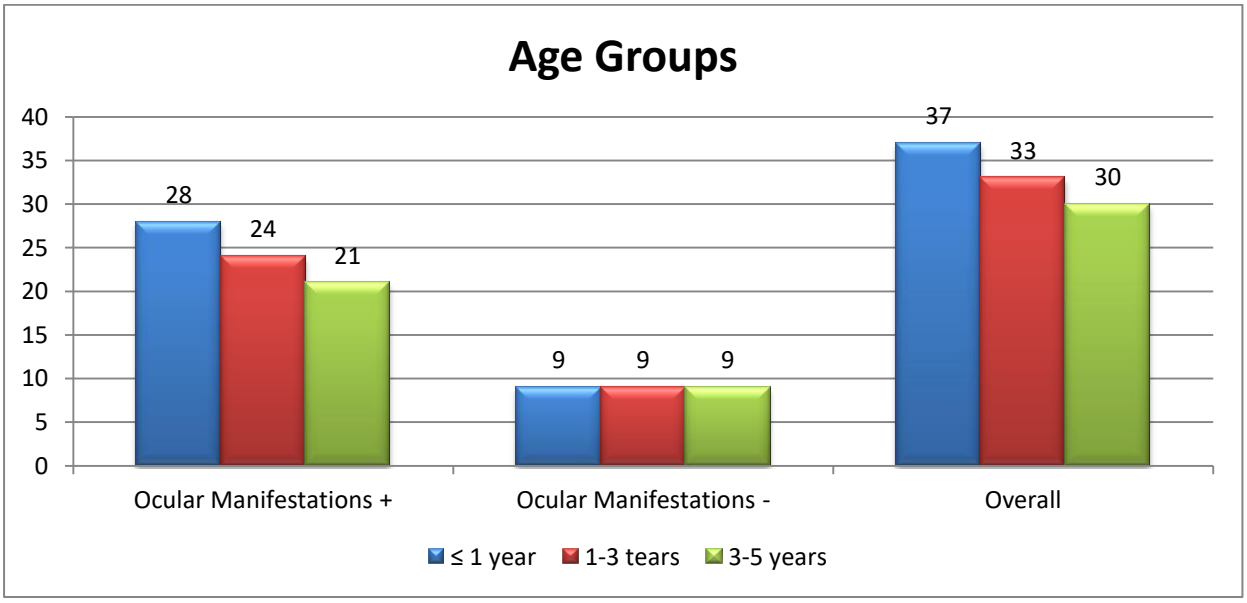
- The second most common manifestation was strabismus (21 cases), with **13** cases having **esotropia** and 7 cases having **exotropia**.
- Other ocular manifestation seen were- **Optic atrophy** (4 cases), **Cataract** (2 cases), **Nystagmus** (3 cases) and **Cortical Visual Impairment** (2 cases).
- The type of cataract seen in both cases was diffuse and the type of nystagmus noted in the 3 cases was horizontal jerky.
- Optic atrophy seen in all 4 cases was primary optic atrophy.



Other Ocular Manifestations	Number	%
Strabismus – Esotropia	13	17.81
Strabismus – Exotropia	7	9.59
Cataract	2	2.74
Nystagmus	3	4.11
Optic Atrophy	4	5.48
Cortical Visual Impairment	2	2.74
Refractive error	42	57.53
Total	73	100.00

AGE

- Out of the **37** children between **6 months to 1 year**, 28 had ocular manifestations, whereas 24 out of the **33** children between **1 year to 3 years** and 21 out of **30** children between **3 to 5 years** had ocular manifestations

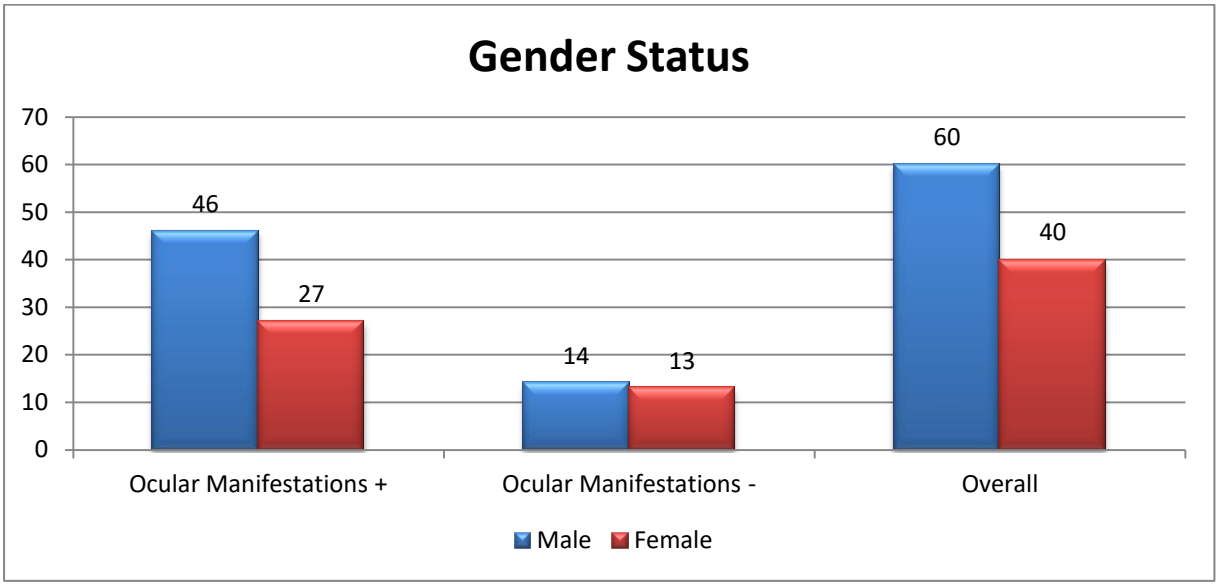


Age Groups	Ocular Manifestations +	%	Ocular Manifestations -	%	Overall	%
≤ 1 year	28	38.36	9	33.33	37	37.00
1-3 tears	24	32.88	9	33.33	33	33.00
3-5 years	21	28.77	9	33.33	30	30.00
Total	73	100.00	27	100.00	100	100.00

Age Distribution	Ocular Manifestations +	Ocular Manifestations -	Overall
Mean	2.21	2.38	2.25
SD	1.45	1.42	1.44
P value Unpaired t Test			0.5943

GENDER

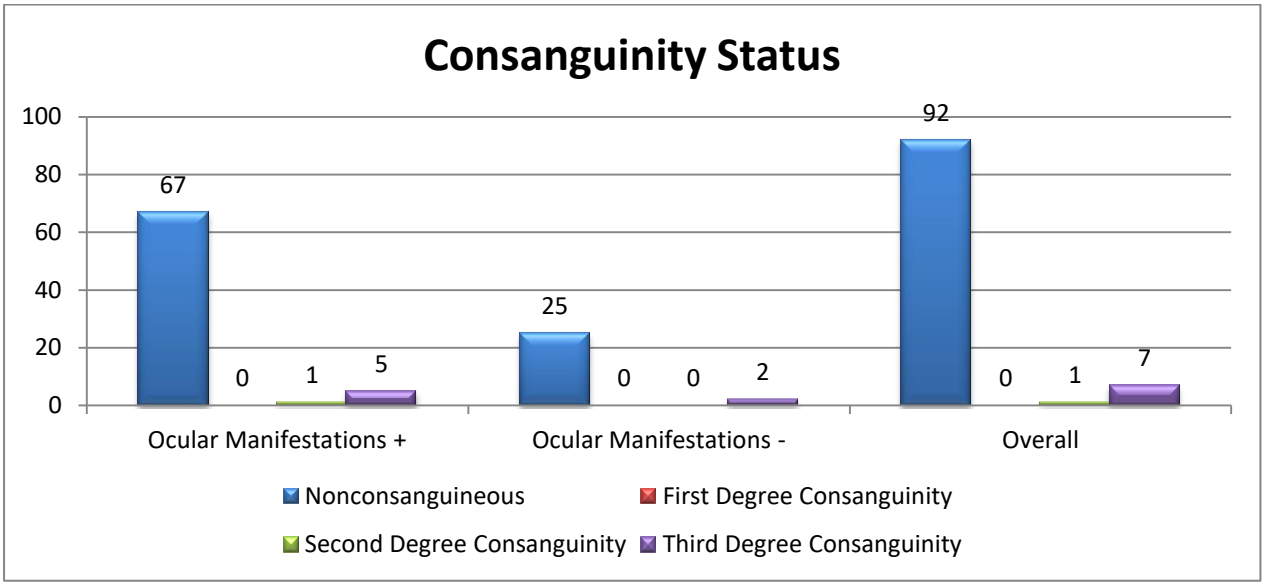
- Out of the 100 children with developmental delay, **60** were **male** out of which **46** had ocular manifestations and **40** were **female** out of which **27** had ocular manifestations.



Gender Status	Ocular Manifestations +	%	Ocular Manifestations -	%	Overall	%
Male	46	63.01	14	51.85	60	60.00
Female	27	36.99	13	48.15	40	40.00
Total	73	100.00	27	100.00	100	100.00
P value Chi Squared Test					0.3166	

CONSANGUINITY

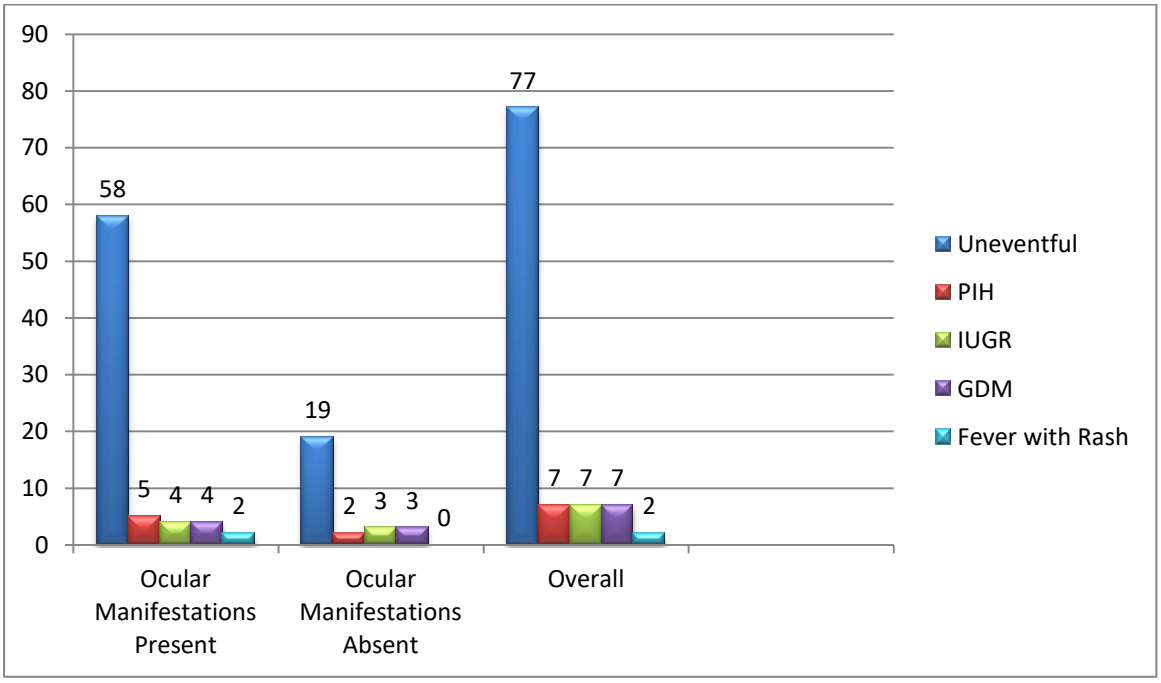
- History of consanguinity was present in **8 cases** (1-second degree and 7-third degree consanguinity), out of which 6 had ocular manifestations



Consanguinity Status	Ocular Manifestations +	%	Ocular Manifestations -	%	Overall	%
Non consanguineous	67	91.78	25	92.59	92	92.00
First Degree Consanguinity	0	0.00	0	0.00	0	0.00
Second Degree Consanguinity	1	1.37	0	0.00	1	1.00
Third Degree Consanguinity	5	6.85	2	7.41	7	7.00
Total	73	100.00	27	100.00	100	100.00
P value Chi Squared Test					0.4211	

ANTENATAL RISK FACTORS

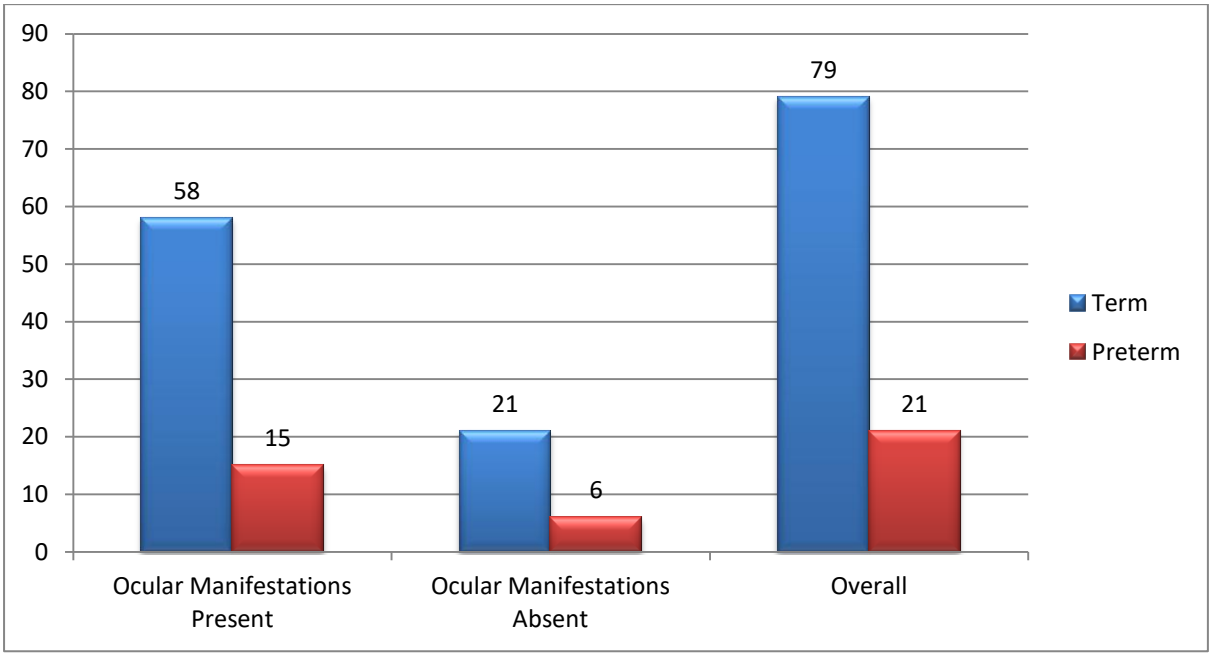
- History of antenatal events was seen in **22 cases** (7-PIH, 7-GDM, 7-IUGR, 2-Fever with rash), out of which **15** children had **ocular manifestations**.



Antenatal Events	Ocular Manifestations +	%	Ocular Manifestations -	%	Overall	%
Uneventful	58	79.45	19	70.37	77	77.00
PIH	5	6.84	2	7.40	7	7.00
IUGR	4	5.47	3	11.11	7	7.00
GDM	4	5.47	3	11.11	7	7.00
Fever With Rash	2	2.74	0	0.00	2	2.00
Total	73	100.00	27	100.00	100	100.00
P value Chi Squared Test					0.3378	

NATAL RISK FACTORS

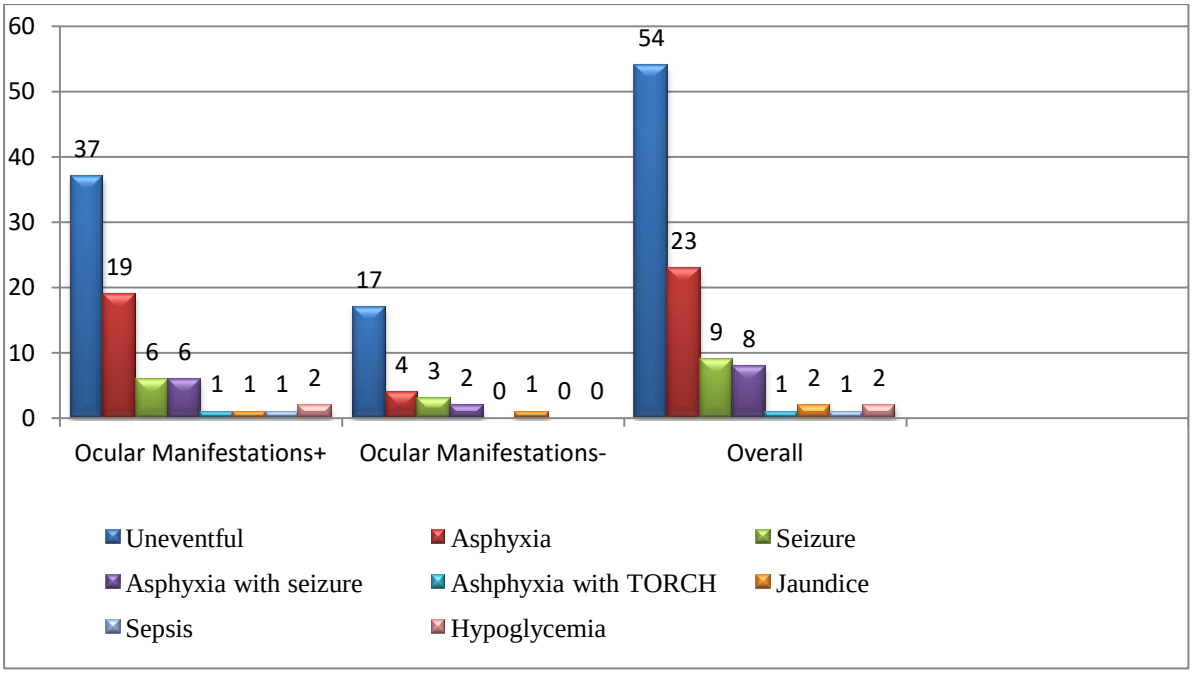
- History of **preterm** delivery was present in **21 cases**, out of which **15** had **ocular manifestations**. 79 cases had history of full-term delivery, out of which 58 had ocular manifestations.



Natal Events	Ocular Manifestations +	%	Ocular Manifestations -	%	Overall	%
Term	58	79.45	21	77.77	79	79.00
Preterm	15	20.54	6	22.22	21	21.00
Total	73	100.00	27	100.00	100	100.00
P value Chi Squared Test					0.4736	

PERINATAL RISK FACTORS

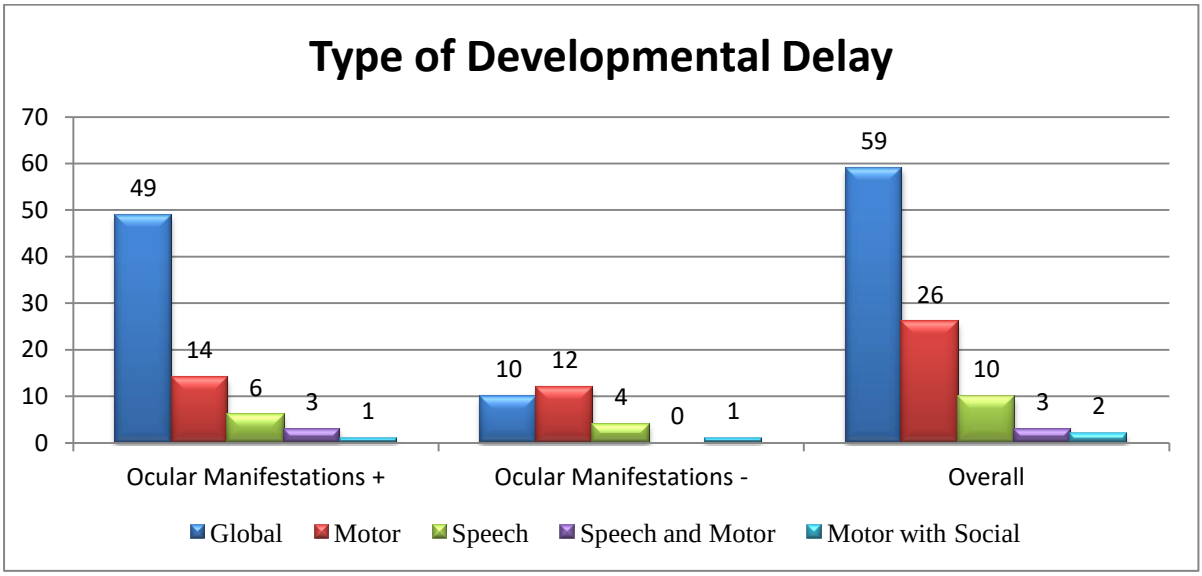
- History of **perinatal events** was positive in **46 children** (Asphyxia-23, Seizures-9, Asphyxia with seizure-8, Asphyxia with TORCH-1, Jaundice-2, Sepsis-1, Hypoglycemia-2), out of which **36** had **ocular manifestations**.



Postnatal Events	Ocular Manifestations +	%	Ocular Manifestations -	%	Overall	%
Uneventful	37	50.68	17	62.96	54	54.00
Asphyxia	19	26.02	4	14.81	23	23.00
Seizures	6	8.21	3	11.11	9	9.00
Asphyxia with seizure	6	8.21	2	7.40	8	8.00
Asphyxia with TORCH	1	1.37	0	0.00	1	1.00
Jaundice	1	1.37	1	3.70	2	2.00
Sepsis	1	1.37	0	0.00	1	1.00
Hypoglycemia	2	2.73	0	0.00	2	2.00
Total	73	100	27	100	100	100
P value Chi Squared Test					0.1993	

TYPE OF DEVELOPMENTAL DELAY

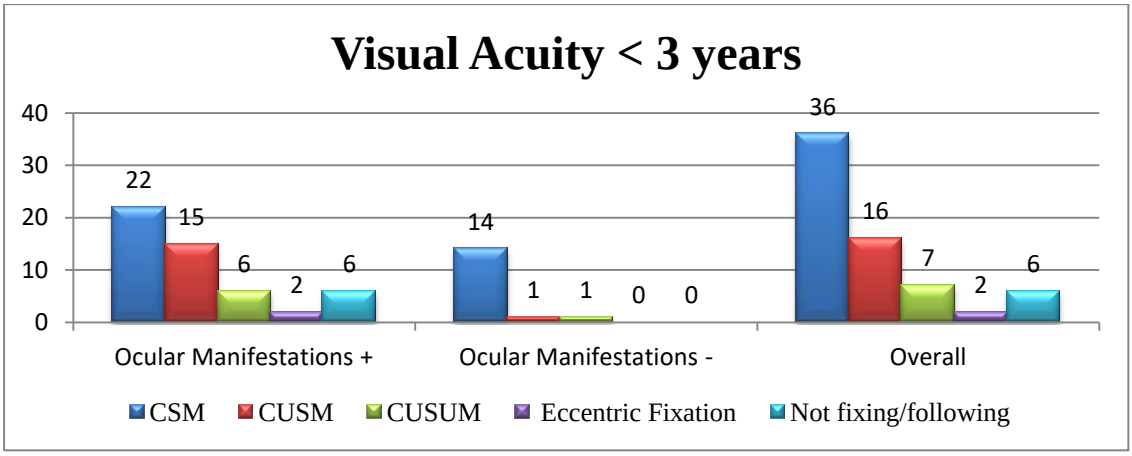
- Among types of developmental delays, the most common was found to be **Global developmental delay** with **59 children** having it, out of which **49** had **ocular manifestations**. The second most common was **motor delays (26 cases),** out of which **14** had ocular manifestations. Other types seen were, speech delays (10 cases), with 6 having manifestations, speech and motor (3 cases) with 3 having manifestations and motor with social delays (2 cases) with 1 having ocular manifestation.



Type of Developmental Delay	Ocular Manifestations +	%	Ocular Manifestations -	%	Overall	%	P value Chi Squared Test
Global	49	67.12	10	37.04	59	59.00	0.0343
Motor	14	19.18	12	44.44	26	26.00	0.0266
Speech	6	8.22	4	14.81	10	10.00	0.6483
Speech and Motor	3	4.11	0	0.00	3	3.00	0.2887
Motor with Social	1	1.37	1	3.70	2	2.00	0.2234
Total	73	100.00	27	100.00	100	100.00	

VISUAL ACUITY < 3 YEARS

- Out of the **36 cases** with **CSM** vision, **22** had **ocular manifestations**.15 out of the **16** cases with **CUSM**, 6 out of the **7** with **CUSUM**, **2** cases with **eccentric fixation** and all **6** of the children **not fixing/following light** had ocular manifestations.

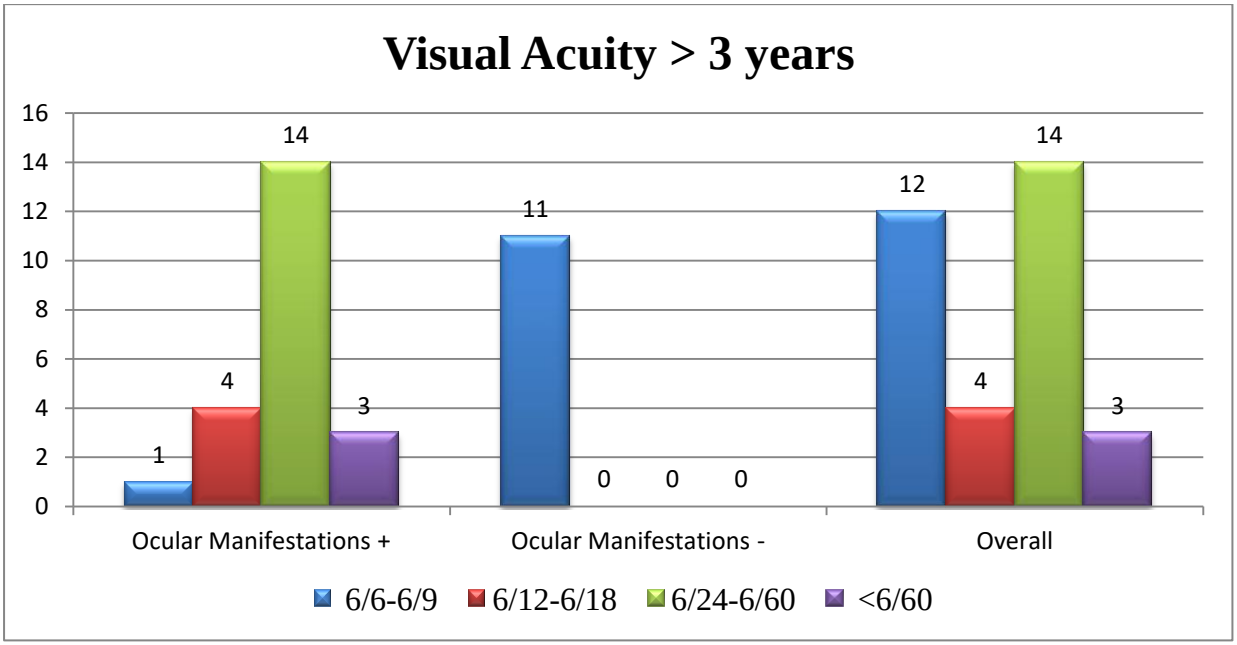


- **CSM=Central Steady Maintained→ 6/6 – 6/9**
- **CSUM=Central Steady Unmaintained→ 6/36 – 6/60**
- **CUSUM=Central Unsteady Unmaintained → <6/60**

Visual Acuity < 3 years	Ocular Manifestations +	%	Ocular Manifestations -	%	Overall	%
CSM	22	43.14	14	87.50	36	53.73
CUSM	15	29.41	1	6.25	16	23.88
CUSUM	6	11.76	1	6.25	7	10.45
Eccentric Fixation	2	3.92	0	0.00	2	2.99
Not fixing/following	6	11.76	0	0.00	6	8.96
Total	51	100.00	16	100.00	67	100.00
P value Chi Squared Test					0.0181	

VISUAL ACUITY > 3 YEARS

- Out of the **12** cases with **6/6-6/9 vision**, only **1** child had **ocular manifestation**. **4** children with **6/12-6/18**, **14** children with **6/24-6/60** and **3** with vision **less than 6/60** had ocular manifestations.



Visual Acuity > 3 years	Ocular Manifestations +	%	Ocular Manifestations -	%	Overall	%
6/6-6/9	1	4.55	11	100.00	12	36.36
6/12-6/18	4	18.18	0	0.00	4	12.12
6/24-6/60	14	63.64	0	0.00	14	42.42
<6/60	3	13.64	0	0.00	3	9.09
Total	22	100.00	11	100.00	33	100.00
P value Chi Squared Test					<0.0001	

RISK FACTORS IN EACH MANIFESTATION:

The risk factors present in the various manifestations were as follows:

Factor	Refractive error(53)	Strabismus(21)	Optic Atrophy(4)	Nystagmus (3)	Cataract(2)	CVI(2)
Consanguinity	**2**	**2**	**-**	**1**	**1**	**1**
Antenatal	**9** (4 PIH, 2 GDM, 1 Fever With Rash, 2 IUGR)	**3** (1 PIH,1 GDM, 1 IUGR)	**2** (1GDM, 1IUGR)	Uneventful	**1** (Fever with rash)	Uneventful
Natal	**8 preterm**	**4 preterm**	**3 preterm**	**1 preterm**	**-**	**2 preterm**
Perinatal	**22** (12 Asphyxia, 4Asphyxia/Seizure, 4 seizure, 1 jaundice, 1hypoglycemia)	**11** (3 Asphyxia,1 TORCH, 3 Seizure, 1 Sepsis, 2Asphyxia/Seizure, 1 Hypoglycemia)	**4** (3Asphyxia, 1 Seizure)	**3** (2 Asphyxia, 1 seizure)	**2** (1Asphyxia, 1 TORCH)	**2** (1 Asphyxia, 1 Seizure)
Type of Developmental delay	GDD-35, Motor-12, Speech-5, Motor & Social-1	GDD-15, Motor-2 , Speech & Motor-2, Speech-2	GDD-4	GDD-2, Motor-1	GDD-1, Motor-1	GDD-2

- Among these, the most significant risk factors were seen in Optic atrophy, Nystagmus , cataract and CVI. Asphyxia and preterm were seen as significant risk factors for optic atrophy and asphyxia was seen as a common risk factor in nystagmus, cataract and CVI.

DISCUSSION

- Our study included 60 male and 40 female children with developmental delay, in the age range of 6 months to 3 years and found a prevalence of ocular manifestations in 73% of the cases, with the most common manifestation being refractive error, followed by strabismus.

- In a study by **Wu H J et al**[30] on 41 children with developmental delay, sex distribution was seen to be 68% males and 32% females. This study showed ocular manifestations in 56.1%. The mean age range included in the study was 3.53+/-2.25 years.

- In both Wu H J et al and another study by Tsai et al, the two most common manifestations seen in these children were strabismus and optic atrophy.

SEX DISTRIBUTION:

Sex Distribution	Our Study	Wu H J et al
Male	60%	68%
Female	40%	32%

OCULAR MANIFESTATIONS:

Ocular Manifestations	Our Study	Wu H J et al	Akinci A et al	Katoch S et al	Charmaine Bridgette Solomon et al	Lagunju et al
Present	73%	56.1%	77%	68%	75.2%	28.2%

- The study conducted by **Akinci A et al**[33] was done in children with intellectual disability out of which 77% had ocular manifestations.
- **Charmaine Bridgette Solomon et al**[32] conducted the study on 125 children with developmental delay, between the age groups of 6 months to 3 years in Calicut.
- **Lagunju et al**[29] studied 149 cerebral palsy children in which prevalence of ocular abnormalities were found to be 28.2%, out of which 61.9% were completely blind.
- Another study conducted by **Katoch S et al**[27] on 200 cases between 8 months to 21 years with cerebral palsy, showed 68% of the cases had visual morbidity.

RISK FACTORS:

Risk factors	Our Study (Overall%)	Charmaine Bridgette Solomon et al
Preterm delivery	20.5%	8.8%
Antenatal factors	23%	20.8%
Perinatal Asphyxia	31%	27%

TYPE OF DEVELOPMENTAL DELAY:

Type of DD	Our Study (Overall %)	Chen et. Al
Global Developmental Delay	59%	58.4%
Speech Delay	10%	16.8%
Motor Delay	26%	4.8%
Others	5%	20%

(Chen et al study was conducted on 1048 children with developmental delay)

REFRACTIVE ERRORS:

Types	Our Study	Katoch S	Elmenshaw	S Nielsen
Refractive errors (Total)	53%	33.5%	71.4%	46.7%
Myopia	10%	13.5%	41.4%	10.8%
Hypermetropia	34%	20%	20%	15.3%
Astigmatism	9%	-	10%	20.6%

- A study by **Banke s et al**[28] included 200 children with developmental delay and concluded that refractive errors were seen in 49%, squint in 37%, nystagmus in 7.5% and other manifestations like cataract, retinopathy of prematurity and optic atrophy.

- In the study conducted by **Elmenshaw[16],** 46 children with cerebral palsy in the age range of 2 years to12 years were examined for ocular abnormalities, which was seen in 35 cases.

Other Manifestations:

Manifestations	Our Study	Banke s et al	Katoch S	Elmenshaw
Strabismus	20%	37%	39%	32.8%
Nystagmus	3%	7.5%	5.5%	11.4%
Optic Atrophy	4%	6.5%	5.5%	20%
Cataract	2%		2.5%	-

- Other than refractive errors, the **Nielson et al study** showed 8.8% optic atrophy and 11.7% cataract. Cortical Visual Impairment (CVI) was seen in 2% of the children in our study whereas in the Nielson study, CVI was seen in 47.05% of children with ocular impairment with perinatal risk factors and in 51.4% of the children in the study conducted by Elmenshaw.

CONCLUSION

- In our study, we have concluded that, 73 out of the 100 children had ocular manifestations.
- The most common ocular manifestation is refractive error (53%), which when treated as early as possible, can prevent the development of amblyopia.
- The second most common manifestation was strabismus (21%), which is again a treatable cause of visual impairment. The one other treatable cause of blindness was cataract, which was seen in 2% of the cases.
- Other manifestations seen were nystagmus (3%), optic atrophy (4%) and CVI (2%).
- Among the children with ocular manifestations, 15 had antenatal events, 15 had history of preterm birth and 36 had history of perinatal risk factors, most common being perinatal asphyxia, which was commonly seen in association with optic atrophy, nystagmus and CVI.
- The most common form of developmental delay was Global developmental delay (59%).
- In a child with developmental delay, it becomes crucial to help the child lead a normal life, personally, socially and academically. Thus it is very important to screen these children for associated ocular abnormalities, when uncorrected, can impede the normal activities of their daily life. It is also important to diagnose the preventable risk factors at an early stage.
- Moreover, as already discussed above, vision in interlinked with other domains of development. Thus the impairment in visual function, can adversely affect their development, causing a further retardation or delay.

- This problem can be averted, if visual screening is carried out and correctable or preventable blindness is managed appropriately. In cases where the visual impairment is not treatable, appropriate management is given, as in cases of amblyopia.
- Thus, a complete ocular examination should become an integral part of clinical work up of all children with delayed milestones, even if there is no evidence of gross ocular dysfunction.
- Early recognition of such abnormalities may prove critical in managing all cases that are amenable to treatment.

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PROFORMA

Serial no. :

Name :

Age :

Sex :

Name of the informant :

Address :

Consanguinity (Yes/No):

Antenatal/Natal History :

Postnatal History :

Developmental History (Social/Gross motor/Fine motor/Language):

Family History :

General Examination:

Ocular examination:

RE **LE**

- Vision
- Eyelids and lashes
- Squint evaluation
- Extraocular movements
- *Slit lamp examination:*
- Conjunctiva
- Cornea
- Anterior chamber
- Iris
- Pupil
- Lens
- Fundus
- Retinoscopy
- Other examinations

CONSENT

நோயாளி தகவல் தாள்

வளர்ச்சி குறைபாடு உள்ள குழந்தைகளில் ஏற்படும் கண் மற்றும் பார்வை குறைபாடுகளை கண்டறியும் ஓர் ஆய்வு.

ஆய்வின் நோக்கம்:

வளர்ச்சி குறைபாடு உள்ள குழந்தைகளில் ஏற்படும் கண் மற்றும் பார்வை குறைபாடுகளை கண்டறியும் ஓர் ஆய்வு.

உண்டாகக் கூடிய இடர்கள்:

அனைத்து முறைகளிலும் இருப்பது போலவே சில எதிர்பாராத இடர்கள் ஏற்படலாம்.

நம்பகத் தன்மை:

உங்கள் குழந்தையின் மருத்துவ பதிவேடுகள் மிகவும் ரகசியமாக வைத்துக் கொள்ளப்படும். மற்ற மருத்துவர்கள், விஞ்ஞானிகள், இந்த ஆராய்ச்சி ஆதரவாளர்களின் பிரதிநிதிகள் ஆகியோரிடமும் அவை வெளிப்படும். இந்த ஆய்வின் முடிவுகள் அறிவியல் பத்திரிக்கைகளில் பிரசுரிக்கப்படலாம்.

ஆய்வில் பங்கேற்கும் நோயாளியின் கடமைப் பொறுப்புகள்:

உங்கள் குழந்தையை கவனித்துக் கொள்ளும் மருத்துவருடன் நீங்கள் முழுமையாக ஒத்துழைக்க வேண்டும் மற்றும் உங்கள் குழந்தையின் மருத்துவரால் குறிப்பிடப்படும் வைத்தியத்தை தவறாமல் பின்பற்ற வேண்டும் என்றும், என்னென்ன செய்யக்கூடாது என்றும் என்னென்ன செய்ய வேண்டும் என்றும் கூறப்பட்டுள்ள வற்றிலிருந்து சற்றும் விலகக்கூடாது என்றும் நீங்கள் எதிர்பார்க்கப்படுகிறீர்கள்.

ஆய்வில் உங்கள் பங்கேற்பு மற்றும் உரிமைகள்:

இந்த ஆய்வில் உங்கள் குழந்தையின் பங்கேற்பு தன்னிச்சையானது. மற்ற காரணங்கள் எதையும் கூறாமலேயே நீங்கள் இந்த ஆய்விலிருந்து உங்கள் குழந்தையை எந்த நேரத்திலும் விலக்கிக் கொள்ளலாம். எந்த ஒரு நேரத்திலும் உங்களுக்கு திருப்தி இல்லை என்று உணர்ந்தாலோ அல்லது வேறு ஏதேனும் உடல் நலக்குறைவு உண்டானாலோ உங்கள் குழந்தையை கவனித்து வரும் மருத்துவரிடம் உடனடியாகத் தெரிவிக்கவும். சிகிச்சை உங்கள் குழந்தைக்கு பொருத்தமாக இருக்காது என்று தோன்றினால் உடனடியாக நிறுத்தப்படும். உங்கள் சம்மதம் இன்றியே கூட ஆய்வு நிறுத்தப்படுவது சாத்தியமே.

KEY TO MASTER CHART

- **Age-** y=Years

  m=Months

- **Sex-**M=Male

  F=Female

- **Consanguinity-**NC=Non Consanguineous

  C=Consanguineous

- **Antenatal-**GDM=Gestational Diabetes Mellitus

  PIH=Pregnancy Induced Hypertension

  IUGR=Intrauterine Growth Retardation

  UE=Uneventful

- **Perinatal-**UE=Uneventful

- **Type of developmental delay-**DD=Developmental delay

  GDD=Global Developmental Delay

  M&S=Motor and Speech

- **Visual Acuity-**CSM=Central Steady Maintained(6/6 – 6/9)

  CSUM=Central Steady Unmaintained (6/36 – 6/60)

  CUSUM=Central Unsteady Unmaintained (<6/60)

- **Other manifestations-** CVI-Cortical Visual Impairment

  B/L- Bilateral

Ocular Manifestations in Developmental Delay- MASTER CHART

S.No	Name	Age/Sex	Consanguinity	Birth History			Type of DD	Visual Acuity		Refractive error	Other manifestations
				Ante natal	Natal	Perinatal		<3 yrs	>3 yrs (Right, Left)		
1.	Md.Thaza	1y/M	NC	UE	Term	UE	Motor	CSM		-	-
2.	Sindhuja	3y/ F	NC	UE	Term	Asphyxia/ Seizure	Motor		6/6, 6/6	-	-
3.	Jeevitha	4y/ F	NC	GDM	Preterm	UE	GDD		6/60, 6/24	Hypermetropia	-
4.	Dharani	7m/ F	NC	PIH	Term	UE	Motor	CSUM		Astigmatism (Hypermetropic)	-
5.	Yuvashree	5y/ F	NC	UE	Term	UE	Motor		6/6, 6/6	-	-
6.	Deepika	9m/ F	NC	UE	Preterm	Asphyxia	GDD	CSM		-	-
7.	Asiya Afreen	2y6m/ F	NC	UE	Term	Asphyxia/ Seizure	GDD	CSM		-	Squint-Esotropia
8.	Fathima	1y6m/ F	NC	UE	Preterm	UE	Motor	CSM		Hypermetropia	-
9.	Mohit	2y/M	NC	GDM	Preterm	Asphyxia	GDD	Not fixing, following		-	Optic Atrophy
10.	Pugazhendi	5y/M	NC	UE	Preterm	Seizure	GDD		6/60, 3/60	Myopia	Squint-Exotropia

S.No	Name	Age/Sex	Consanguinity	Birth History			Type of DD	Visual Acuity		Refractive error	Other manifestations
				Ante natal	Natal	Perinatal		<3 yrs	>3 yrs		
11.	Ehilarasi	1y/ F	NC	PIH	Term	Jaundice	GDD	CSM		-	-
12.	Jeevan	11m/M	NC	UE	Term	Asphyxia	GDD	CSM		Astigmatism (Hypermetropic)	-
13.	Mohanapriya	2y/M	NC	IUGR	Term	UE	GDD	CSUM		Myopia	-
14.	Mesaiah	4 y/M	NC	IUGR	Preterm	UE	GDD		6/6, 6/6	-	-
15.	Christina	9m/ F	NC	UE	Term	Asphyxia	Motor	CSM		-	-
16.	Rasikashree	3 y6m/ F	NC	UE	Term	Sepsis	M&S	CSUM		-	Squint-Exotropia
17.	Md.Thousif	8m/M	NC	UE	Term	UE	Motor	CSM		Hypermetropia	-
18.	Antony Jerald	1y/M	NC	UE	Term	Hypoglycemia	Speech	CSM		Hypermetropia	-
19.	Augustin	3y/M	C-III degree	IUGR	Preterm	UE	GDD	CUSUM		-	-
20.	Shivanya	7m/ F	NC	UE	Term	Asphyxia/Seizure	GDD	CSM		Myopia	-
21.	Arivazhagi	2y/ F	NC	UE	Term	UE	GDD	CSUM		Hypermetropia	-
22.	Manikandan	7m/M	C-III degree	Fever with rash	Term	Asphyxia	GDD	Not fixing, following		-	B/L Cataract

S.No	Name	Age/Sex	Consanguinity	Birth History			Type of DD	Visual Acuity		Refractive error	Other manifestations
				Ante natal	Natal	Perinatal		<3 yrs	>3 yrs		
23.	Abuhuraira	3y/M	NC	UE	Preterm	UE	GDD		6/24, 6/36	Hypermtetropia	-
24.	Gayathri	1y4m/ F	NC	UE	Term	Seizures	Speech	CSUM		Astigmatism (Myopic)	-
25.	Dinesh	7m/M	NC	UE	Term	Asphyxia	GDD	CUSUM		Hypermetropia	Nystagmus
26.	Kiran	6m/M	NC	UE	Preterm	Asphyxia /Seizures	GDD	Not fixing, following		-	CVI
27.	Sharanya	1y/ F	NC	UE	Term	UE	Speech	CSM		-	Squint-Esotropia
28.	Kamalakannan	4y/M	NC	PIH	Term	UE	M&S		6/9, 6/12	-	Squint-Exotropia
29.	Joseph	3y7m/M	NC	UE	Term	UE	GDD		6/24, 6/36	Hypermetropia	-
30.	Mani	7m/M	NC	UE	Term	Asphyxia	GDD	CSM		-	-
31.	Sai preethika	1Y3M	C-II degree	UE	Preterm	Asphyxia	Motor	CUSUM		Hypermetropia	Nystagmus
32.	Ananya	1Y/ F	NC	UE	Term	Seizure	GDD	Not fixing, following		-	Optic Atrophy/ Nystagmus
33.	Divya	7m/ F	NC	UE	Term	UE	GDD	CSM		-	Squint-Esotropia
34.	Barghav	6m/M	NC	UE	Term	UE	GDD	CSM		Hypermetropia	-

S.No	Name	Age/Sex	Consanguinity	Birth History			Type of DD	Visual Acuity		Refractive error	Other manifestations
				Ante natal	Natal	Perinatal		<3 yrs	>3 yrs		
35.	Adhithya	5y/M	NC	UE	Term	UE	GDD		6/18, 6/24	Myopia	-
36.	Jayashri	8m/ F	NC	UE	Term	UE	GDD	Eccentric fixation		Hypermetropia	Squint-Esotropia
37.	Amish	2Y6M/M	NC	UE	Term	UE	Speech	CSUM		Myopia	-
38.	Jyoti	9m/ F	NC	UE	Term	Seizure	GDD	CSM		-	-
39.	Muhammed Musahir	4y6m/M	NC	UE	Term	Asphyxia/ Seizure	GDD		6/24, 6/18	Astigmatism (Mixed)	Squint-Esotropia
40.	Rohit	11m/M	NC	UE	Term	Asphyxia/ Seizure	GDD	CSUM		Hypermetropia	-
41.	Lavanya	2Y2m/F	NC	UE	Term	Asphyxia/ TORCH	Motor	Eccentric fixation		-	(L) Cataract/ Exotropia
42.	Shanmugaraj	1y8m/M	NC	GDM	Preterm	UE	Motor	CSM		-	-
43.	Keerthi	10m/M	NC	UE	Term	Seizures	GDD	CSM		-	-
44.	Jamila	3y7m/ F	NC	IUGR	Preterm	Asphyxia	GDD		1/60, 2/60	-	Optic Atrophy
45.	Karthik	4y/M	NC	UE	Preterm	UE	Motor		6/6, 6/6	-	-
46.	Geethapriya	1y/ F	NC	UE	Term	Asphyxia	GDD	CSM		Hypermetropia	-
47.	Kalaivani	4y/ F	NC	PIH	Term	UE	Motor		6/24, 6/36	Hypermetropia	-
48.	Jeeva	5y/M	C-III degree	UE	Term	UE	GDD		6/6, 6/6	-	Squint-Esotropia

S.No	Name	Age/Sex	Consanguinity	Birth History			Type of DD	Visual Acuity		Refractive error	Other manifestations
				Ante natal	Natal	Perinatal		<3 yrs	>3 yrs		
49.	Yogesh	3y5m/M	NC	UE	Term	UE	Speech		6/12, 6/12	Myopia	Squint-Exotropia
50.	Varun	4y/M	NC	PIH	Term	UE	Speech		6/6, 6/6	-	-
51.	Alex	3Y/M	NC	UE	Preterm	UE	GDD	CUSUM		Hypermetropia	Squint-Esotropia
52.	Ambreen	2y/ F	NC	UE	Term	Asphyxia/ Seizure	Social & Motor	CSM		-	-
53.	Ajay	3y8m/M	NC	UE	Term	Asphyxia	GDD		6/12, 6/18	Hypermetropia	-
54.	Mourish	4y/M	NC	UE	Term	Asphyxia	GDD		6/60, 6/36	Myopia	Squint-Exotropia
55.	Sai Prithvi	11m/M	NC	UE	Term	Asphyxia/ Seizure	GDD	CSM		Hypermetropia	-
56.	Ahalya	5y/ F	NC	UE	Term	UE	Social & Motor		6/60 , 6/24	Myopia	-
57.	Siva	2Y/M	NC	UE	Term	Asphyxia	GDD	CSUM		Hypermetropia	Squint-Esotropia
58.	Vijayan	9m/M	NC	UE	Preterm	Asphyxia	GDD	Not fixing, following		-	Optic Atrophy
59.	Jhanvi	1y/ F	NC	UE	Term	Asphyxia	GDD	CSM		Astigmatism (Myopic)	-
60.	Mahesh	1y6m/M	NC	UE	Term	UE	Motor	CSUM		Astigmatism (Myopic)	-
61.	Abishek	2y6m/M	NC	IUGR	Preterm	UE	Speech	CSM		-	-

S.No	Name	Age/Sex	Consanguinity	Birth History			Type of DD	Visual Acuity		Refractive error	Other manifestations
				Ante natal	Natal	Perinatal		<3 yrs	>3 yrs		
62.	Balaji	2y6m/M	NC	IUGR	Term	UE	GDD	CSUM		Hypermetropia	-
63.	Shravan	3y/M	NC	UE	Term	Asphyxia	GDD		6/60, 6/24	Astigmatism (Hypermetropic)	-
64.	Paul sankar	1y7m/M	NC	UE	Preterm	UE	GDD	CSM		Hypermetropia	-
65.	Boopathi	2y/M	NC	UE	Term	UE	GDD	CSUM		Hypermetropia	-
66.	Anju	1y7m/ F	NC	UE	Preterm	Hypoglycemia	GDD	CSM		-	Squint-Esotropia
67.	Devnath	2y/M	NC	UE	Term	UE	GDD	CSM		Hypermetropia	-
68.	Sourav	2y6m/M	C-III degree	UE	Preterm	Asphyxia	GDD	Not fixing, following		-	CVI
69.	Kavin	4y/M	NC	UE	Term	UE	Motor		6/12, 6/12	Astigmatism (Myopic)	-
70.	Kavya	4y/ F	NC	Fever with rash	Term	Asphyxia	GDD		6/60, 6/24	Hypermetropia	-
71.	Pradeep	3y/M	NC	GDM	Term	Asphyxia	M&S	CUSUM		-	Nystagmus
72.	Katheeja	11m/ F	NC	PIH	Term	UE	Speech	CSM		Astigmatism (Myopic)	-
73.	Md.Nawaz	10m/M	NC	UE	Term	Asphyxia	Motor	CUSUM		Myopia	-

S.No	Name	Age/Sex	Consanguinity	Birth History			Type of DD	Visual Acuity		Refractive error	Other manifestations
				Ante natal	Natal	Perinatal		<3 yrs	>3 yrs		
74.	Deepak	2y/ M	NC	UE	Term	UE	GDD	CSUM		Hypermetropia	-
75.	Farhan	1y6m/M	NC	UE	Term	Asphyxia	GDD	CSM		-	-
76.	Marjiya	7m/F	NC	UE	Term	UE	Motor	CSM		-	
77.	Adharsh	9m/M	NC	UE	Term	Seizure	GDD	CSM		Hypermetropia	-
78.	Sujitha	2y/F	C-III degree	UE	Term	UE	Motor	CSM		-	-
79.	Sidarth	9m/M	NC	GDM	Term	Seizure	GDD	CSUM		Hypermetropia	Squint-Esotropia
80.	Nikitha	3y/F	NC	UE	Term	UE	GDD		6/6, 6/6	-	-
81.	Sangeetha	4y/F	NC	UE	Term	UE	Motor		6/6, 6/6	-	-
82.	Gautham	1y/M	NC	UE	Term	UE	Motor	CSM		-	-
83.	Balaguru	1y6m/M	NC	UE	Term	Asphyxia	GDD	CSM		Hypermetropia	-
84.	Ankitha	11m/F	NC	UE	Term	Asphyxia	GDD	CSUM		-	Squint-Exotropia
85.	Anand	3y6m/M	NC	UE	Term	UE	Motor		6/6, 6/6	-	-
86.	Priya	2y6m/F	NC	UE	Term	UE	Motor	CSUM		Hypermetropia	-
87.	Vikram	6m/M	NC	UE	Term	Seizure	GDD	CSUM		-	Squint-Esotropia
88.	Narayan	1y6m/M	NC	UE	Term	UE	GDD	CSM		Hypermetropia	-

S.No	Name	Age/Sex	Consanguinity	Birth History			Type of DD	Visual Acuity		Refractive error	Other manifestations
				Ante natal	Natal	Perinatal		<3 yrs	>3 yrs		
89.	Julia	4y/F	NC	GDM	Term	UE	Speech		6/6,6/6	-	-
90.	Ramgopal	1y/M	NC	IUGR	Term	UE	GDD	CSM		-	Squint-Esotropia
91.	Oviya	5y/F	C-III degree	UE	Term	UE	Motor		6/24, 6/24	Hypermetropia	-
92.	Nithish	7m/M	NC	UE	Term	UE	GDD	CSM		Hypermetropia	-
93.	Helen	3y6m/F	NC	UE	Term	Seizure	Speech		6/6, 6/6	-	-
94.	Jagadish	4y/M	NC	UE	Term	Jaundice	Motor		6/24, 6/18	Hypermetropia	-
95.	Bhargavi	3y8m/F	NC	UE	Term	UE	GDD		6/60, 6/24	Myopia	-
96.	Vanathi	10m/F	NC	UE	Term	UE	Motor	CUSUM		-	Squint-Exotropia
97.	Vasu	4y/M	NC	UE	Term	UE	GDD		6/36, 6/60	Hypermetropia	-
98.	Jayanth	4y6m/M	NC	GDM	Term	UE	Motor		6/6, 6/6	-	-
99.	Krishna	5y/M	NC	PIH	Preterm	UE	Motor		6/60, 6/36	Hypermetropia	-
100.	Amudha	7m/F	C-III degree	UE	Preterm	UE	GDD	CSM		-	Squint-Esotropia